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VETERINARY STUDENT ENROLLMENT FOR 1938-1939

Although the trend in the enrollment of veterinary students continues upward, the increase shown this year is the smallest in five years. This year, the 14th consecutive year to show an increase, the gain is only 195 over last year, as compared with an average gain of 305 each year for the four years previous. However, in these comparisons, it should be noted that Kansas State College no longer reports pre-veterinary students.

It has been suggested by several of the deans that pre-veterinary students should be left out of consideration in these reports. The point is well taken, but, unfortunately, we started including them in the report for the year 1932-33. For the sake of being able to make comparisons from year to year, we have continued to include figures on pre-veterinary students. These are presented in a way, however, that makes it easy to segregate them.

Cornell University, the University of Pennsylvania, and the two Canadian veterinary colleges do not report pre-veterinary students. Then, as has been pointed out before, our figures on pre-veterinary students cover only those registered in institutions having courses in veterinary medicine. There undoubtedly are scores of students preparing for veterinary medicine besides those accounted for at Alabama, Colorado, Iowa, Michigan, Ohio, Texas and Washington.

For the purpose of making some comparisons on veterinary student enrollment from year to year, with pre-veterinary students left out of consideration, let us look at the figures in table I. These rep-

TABLE I—*Enrollment of regular veterinary students for years 1929-30 to 1938-1939.*

COLLEGE YEAR	STUDENTS
1929–1930	1053
1930–1931	1212
1931–1932	1378
1932–1933	1401
1933–1934	1370
1934–1935	1568
1935–1936	1810
1936–1937	1927
1937–1938	2170
1938–1939	2339

resent the enrollment of regular students (freshmen, sophomores, juniors and seniors) for the past ten years. We find that there has been an increase every year but one (1933-34) and that the total enrollment has more than doubled during the ten-year period. It has more than trebled since 1927-28 and has almost quadrupled since 1924-25. This year there are more freshman students enrolled than there were students in all classes any year during the period 1923-27.

At no time in the past has veterinary education in the United States received the attention that is being given to it at present. Some of the questions that are being debated are the following:

1. Are more veterinary colleges needed?
2. Are our present veterinary colleges properly located geographically and economically?
3. What is to become of the colleges that are located in states that are unable to support them adequately?
4. Is direct federal financial support a desirable solution of the present problem?
5. Where are our colleges going to secure capable teachers for the present large number of veterinary students?
6. What can be done to stimulate graduate work in veterinary medicine?
7. Is the country in need of more veterinarians?
8. Is it desirable to lengthen the veterinary curriculum another year?
9. Should a year of interneship be required of all graduates?
10. What changes should be made in the present curriculum with a view to turning out better prepared veterinarians?

We are going to ask some of our leaders in veterinary education to answer these questions in the JOURNAL. Watch for the answers.

Table II shows the total enrollment of veterinary students in the ten colleges in the United States and the two in Canada, for the current year.

TABLE II—*Veterinary student enrollment for the college year 1938-1939.*

COLLEGE	FRESH.	SOPH.	JUN.	SEN.	SPEC.	GRAD.	TOTAL	1937–38	CHANGE
Alabama Polytechnic Institute	71	52	46	29	69*	0	267	214	+53
Colorado State College	41	37	38	28	58*	0	202	195	+7
Cornell University	41	38	41	40	0	9	169	165	+4
Iowa State College	63	63	55	50	128*	7	366	299	+67
Kansas State College	62	52	57	68	0	2	241	282	—41
Michigan State College	56	64	58	36	77†	6	297	254	+43
Montréal, Université de	15	11	14	13	0	0	53	53	0
Ohio State University	70	61	48	48	88*	9	324	301	+23
Ontario Veterinary College	68	68	61	62	0	0	259	241	+18
Pennsylvania, University of	51	49	36	52	8	0	196	189	+7
Texas A. and M. College	101	84	41	61	67†	3	357	340	+17
Washington, State College of	41	38	32	28	97*	1	237	239	—2
Totals (1938-39)	680	617	527	515	592	37	2968	2772	+196
Last year (1937-38)	657	528	542	443	564	38	2772	2501‡	+271

*All pre-veterinary students.
†All but one are pre-veterinary students.
‡Total number of students for 1936-37.

COMMITTEE ON RABIES

President Bergman has appointed the Special Committee on Rabies, authorized by the A. V. M. A. at the meeting in New York. The personnel of the Committee is as follows:

Lt. Col. R. A. Kelser, *Chairman*, Office of the Surgeon General, Washington, D. C.
Dr. M. F. Barnes, Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.
Dr. Geo. E. Corwin, Deputy Commissioner on Domestic Animals, Hartford, Conn.
Dr. C. E. Cotton, Minnesota State Live Stock Sanitary Board, Saint Paul, Minn.
Dr. L. M. Hurt, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

A change of fortune hurts a wise man no more than a change of the moon.—Benjamin Franklin.

EQUINE ENCEPHALOMYELITIS LINKED WITH HUMAN ENCEPHALITIS

During August, equine encephalomyelitis made its appearance in southwestern Massachusetts. At about the same time, some 20 cases of encephalitis in children occurred in the same territory, with a mortality rate of about 50 per cent.

In September, Fothergill, Dingle, Farber and Connerley[1] reported the isolation of eastern equine encephalomyelitis virus from a fatal human case. Lt. Col. R. A. Kelser, of the Army Veterinary Corps, was called to Boston in consultation and returned to Washington with samples of the human virus, which he turned over to Dr. H. W. Schoening, of the U. S. Bureau of Animal Industry. The latter inoculated horses with some of the virus and ran animal-protection tests, the results of which fully substantiated the findings of Fothergill and his associates.[2] Later, Webster and Wright,[3] of the Rockefeller Institute, confirmed these findings by means of animal inoculations and specific neutralization tests with hyperimmune equine encephalomyelitis serum.

According to information available, none of the human cases were in contact with affected horses, nor were they in contact with one another. All of the evidence indicated mosquito transmission and prophylactic measures adopted by the Massachusetts Department of Health were based on the theory of mosquito transmission.

It is believed that this is the first time that the disease known as equine encephalomyelitis has been definitely linked with a similar disease in man.

ANOTHER A. V. M. A. PAST PRESIDENT PASSES

It is with a heavy heart that we pen these last few lines for this issue of the JOURNAL. A telegram from Philadelphia has just brought the word of the death of Dr. C. J. Marshall, for over 40 years a member of the veterinary faculty of the University of Pennsylvania. As a student, practitioner, teacher, writer, state veterinarian, past secretary and past president of the A. V. M. A., he was known to thousands of veterinarians in all parts of the world. Those who knew him best, however, were those thrice fortunate ones whose privilege it was to sit in his classes and know him as the teacher, or to accompany him to stable or farm and know him as the clinician. He has gone to join that trio of masters, Pearson, Adams and Harger, with whom he labored for so many years.

[1]Fothergill, L. D., Dingle, J. H., Farber, S., and Connerley, M. L.: New Eng. Jour. Med., Sept. 22, 1938.
[2]Personal communication.
[3]Webster, L. T., and Wright, F. H.: Sci., lxxxviii (1938), pp. 305-306.

New England Meeting Postponed

The annual meeting of the New England Veterinary Medical Association, which was scheduled to be held in Concord, N. H., on October 3-4, had to be postponed on account of the conditions which existed in that part of the country following the hurricane and floods. The officers decided to hold a one-day meeting in Boston on October 26. We hope to be able to publish a report of the meeting.

Deaths from Cancer Increase

The death rate from cancer continues to increase, in the face of a decrease in the death rate from all causes, according to statistics compiled by the U. S. Public Health Service, covering the first six months of 1938. The infant mortality rate and the percentage of deaths from tuberculosis and influenza-pneumonia declined.

There was a slight increase in the birth rate during the six-month period, as compared with the rate for 1937.

Rabies Costly to Ingham County

The Ingham County (Mich.) Health Department reports that Pasteur treatments of persons exposed to rabies in the county outside the city of Lansing, during the recent outbreak of the disease, will cost approximately $1,500.00. From May 1 to the latter part of September, 92 persons were exposed and received treatment.

APPLICATIONS FOR MEMBERSHIP

This month we are listing 27 applications for membership for the first time and giving the usual second listing to 15 applications. On reviewing the number of applications listed since the New York meeting, we find that 1938 is somewhat behind recent years for the same period.

In August, we listed 51 applications, compared with a three-year (1935-36-37) average of 43 for this month. In September, we listed eight applications, as against the 1935-36-37 average of 54 for this month. In October, 15 applications were listed, compared with 18 as the average for the same month in 1935, 1936 and 1937. The number listed this month compares favorably with the average of 26 for the previous three-year average.

Summarizing the above figures, we find as follows:

Month	*1935*	*1936*	*1937*	*1938*
August	35	27	66	51
September	38	45	80	8
October	16	25	12	15
November	15	50	12	27
Totals.....	104	147	170	101

What do these figures mean? Simply that we are not receiving as many applications this year as we did in 1935, 1936 and 1937, when, as a matter of fact, we should be receiving more. Of the 429 graduates in the class of 1938, only 56 have filed applications, less than 14 per cent. Next month we will present more detailed information on this phase of the situation.

FIRST LISTING

(See July, 1938, JOURNAL)

AL'EN, GAYLE D.
507 Federal Bldg., Little Rock, Ark.
D. V. M., Kansas State College, 1933. Vouchers: Allen W. Rice and Fred M. Shigley.

ALLEN, RICHARD K.
Box 175, Woodland, Calif.
D. V. M., Texas A. and M. College, 1936. Vouchers: W. L. Curtis and A. G. Feers.

BAKER, CHARLES L.
2016 W. 18th St., Little Rock, Ark.
M. D. V., McKillip Veterinary College, 1911. Vouchers: C. D. Stubbs and Allen W. Rice.

BARRETT, JOHN H.
1 Newton Ave., Westerly, R. I.
D. V. M., Cornell University, 1930. Vouchers: J. P. Delaplane and James W. Armstrong.

BENN, ROBERT K.
507 Federal Bldg., Little Rock, Ark.
D. V. M., Kansas City Veterinary College, 1914. Vouchers: C. D. Stubbs and Allen W. Rice.

BRADSHAW, H. D.
3550 Douglas Ave., Memphis, Tenn.
D. V. M., Alabama Polytechnic Institute, 1927. Vouchers: John H. Gillmann and C. P. Branigan.

DENMAN, C. W.
Hughes, Ark.
D. V. M., Kansas City Veterinary College, 1916. Vouchers: John H. Gillmann and C. D. Stubbs.

EDGAR, JEAN R.
323 Chester Ave., Bakersfield, Calif.
B. S., D. V. M., State College of Washington, 1926. Vouchers: P. R. Edwards and Frank Edwards.

ELANDER, BURMAN J.
2905 El Camino Real, San Mateo, Calif.
B. S., D. V. M., State College of Washington, 1938. Vouchers: E. E. Wegner and J. K. Perry.

FOLINSBEE, J. A.
9911-114 St, Edmonton, Alta., Can.
B. V. Sc., University of Toronto, 1937. Vouchers: J. H. Swail and J. C. Hargrave.

GARNER, PAUL C.
Chetek, Wis.
D. V. M., Iowa State College, 1937. Vouchers: H. D. Bergman and James S. Healy.

GARRETT, FRANCIS O., JR.
Dumas, Ark.
D. V. M., Alabama Polytechnic Institute, 1938. Vouchers: C. D. Stubbs and Rease Mitcham.

GREEN, T. C.
1500 Jefferson St., Bluefield, W. Va.
D. V. M., Indiana Veterinary College, 1911. Vouchers: Leslie F. Thayer and H. M. Newton.

MERRILL, CHESTER M.
High St., South Paris, Maine.
D. V. M., U. S. College of Veterinary Surgeons, 1913. Vouchers: C. F. Davis and J. F. Witter.

MITCHELL, GEORGE E.
350 Federal Bldg., Memphis, Tenn.
D. V. M., Cincinnati Veterinary College, 1915.
Vouchers: C. W. Turrell and John H. Gillmann.

MOORE, GEORGE R.
Kansas State College, Manhattan, Kan.
B. S., Central State Teachers' College; D. V. M., Michigan State College, 1938.
Vouchers: Edwin J. Frick and R. R. Dykstra.

OLSON, NORMAN O.
Box 332, Preston, Idaho.
B. S., D. V. M., State College of Washington, 1938. Vouchers: E. E. Wegner and E. T. Baker.

ROBERTS, STEPHEN J.
Kansas State College, Manhattan, Kan.
D. V. M., Cornell University, 1938. Vouchers: Edwin J. Frick and R. R. Dykstra.

SCHOOLEY, MAURICE
Kinston, N. C.
D. V. M., Kansas State College, 1938. Vouchers: Edwin J. Frick and Wm. Moore.

SHORT, ROBERT W.
2116 E. Colorado St., Pasadena, Calif.
D. V. M., Iowa State College, 1938. Vouchers: W. L. Curtis and G. W. Blanche.

TAYLOR, ALBERT A.
2620 Bellevue Ave., Los Angeles, Calif.
B. S., D. V. M., State College of Washington, 1937. Vouchers: W. L. Curtis and Lt. Col. C. E. Pickering.

THOMPSON, FRED
717 State St., Little Rock, Ark.
D. V. M., Alabama Polytechnic Institute, 1937. Vouchers: C. D. Stubbs and Rease Mitcham.

TRAYLOR, DAVID H.
420 Edgewood Ave. N. E., Atlanta, Ga.
D. V. M., Alabama Polytechnic Institute, 1938. Vouchers: Chas. C. Rife and J. E. Severin.

VIETTI, JOHN D.
107 12th St., Puyallup, Wash.
B. S., University of California; B. S., D. V. M., State College of Washington, 1938. Vouchers: Robert R. Weller and C. E. Sawyer.

WARD, BENJAMIN F.
680 Jordan St., Shreveport, La.
D. V. M., Iowa State College, 1938. Vouchers: Col. B. A. Seeley and L. A. Mosher.

WATSON, JOHN W.
6 E. 9th St., New York, N. Y.
V. S., Ontario Veterinary College, 1892.
Vouchers: C. W. Crowley and J. E. Sherwood.

WRIGHT, DONALD A.
Fowler, Mich.
D. V. M., Michigan State College, 1938.
Vouchers: C. F. Clark and B. J. Killham.

The amount which should accompany an application filed this month is $5.83, which covers membership fee and dues to January 1, 1939, including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.83, the additional $5.00 being for the 1939 dues.

Applications Pending

SECOND LISTING

(See October, 1938, JOURNAL)

Akins, Everett H., 6825 Pearl St., Jacksonville, Fla.

Anderson, Dan J., 1619 S. Laredo St., San Antonio, Texas.

Brillhart, Carl E., Apt. 4, 1124 S. 31st St., Omaha, Neb.

Dow, H. G., 1030 Ave. C., Fort Madison, Iowa.

Edgett, James A., 379 New Park Ave., Hartford, Conn.

Gould, Clinton L., Fort Missoula, Mont.

Harkavy, H. J., Box 234, Nowata, Okla.

Levine, P. P., New York State Veterinary College, Ithaca, N. Y.

Rawls, Benjamin H., Box 804, Daytona Beach, Fla.

Sampson, Jesse, University of Illinois, Urbana, Ill.

Sarsohn, J. Jewell, 5791 Westminster Place, Saint Louis, Mo.

Stephens, Clayton, Tupelo, Miss.

Sutherland, John Wm., Pembroke, West, Bermuda.

Welnhofer, John, 2954 N. Menard Ave., Chicago, Ill.

Wood, Armour C., 2031 S. Broad St., Trenton, N. J.

State Board Examinations

Nebraska Veterinary Medical Examining Board. State Capitol, Lincoln, Neb. November 8-9, 1938. Applications must be on file 15 days before date of first examination. Further information may be obtained from Mrs. Clark Perkins, Bureau of Examining Boards, State Capitol, Lincoln, Neb.

Connecticut Board of Veterinary Registration and Examination. January 3, 1939. Further information may be obtained from Dr. Geo. E. Corwin, Secretary, State Capitol, Hartford, Conn.

Poultry Science Research Prize

Doctors Erwin Jungherr and Walter Landauer have been awarded the "Poultry Science Research Prize for 1938," of $100.00 for their paper, "A Condition Resembling Osteopetrosis (Marble Bone) in the Common Fowl," published as Storrs (Conn.) Agricultural Experiment Station Bulletin 222.

COMING VETERINARY MEETINGS

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. November 1, 1938. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Connecticut Veterinary Medical Association. Waterbury, Conn. November 2, 1938. Dr. Geo. E. Corwin, Secretary, State Office Building, Hartford, Conn.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. November 2, 1938. Dr. J. B. Engle, Box 432, Summit, N. J.

Central New York Veterinary Medical Association. Onondaga Hotel, Syracuse, N. Y. November 3, 1938. Dr. W. B. Switzer, Secretary, Route 5, Oswego, N. Y.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. November 3, 1938. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. November 3, 1938. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Southern States Veterinary Medical Association. November 3-5, 1938. Andrew Johnson Hotel, Knoxville, Tenn. Dr. L. A. Mosher, Secretary, Box 1533, Atlanta, Ga.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. November 8, 1938. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. November 9, 1938. Dr. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. November 9, 1938. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Midwest Small Animal Association. Burlington Hotel, Burlington, Iowa. November 10, 1938. Dr. C. L. McGinnis, Secretary, 1314 Main St., Peoria, Ill.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. November 16, 1938. Dr. B. B. Coale, Secretary, 203 Administration Building, Union Stock Yards, Los Angeles, Calif.

Kansas City Veterinary Medical Association. Fort Leavenworth, Kans. November 19, 1938. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Laboratories, Balboa Park, San Diego, Calif. November 21, 1938. Dr. Glenn A. Tucker, Secretary, Vista, Calif.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. November 23, 1938. Dr. C. S. Rockwell, Secretary 4927 Osage Ave., Philadelphia, Pa.

Horse and Mule Association of America. Palmer House, Chicago, Ill. November 30, 1938. Wayne Dinsmore, Secretary, 407 S. Dearborn St., Chicago, Ill.

National Association of B. A. I. Veterinarians. Hotel La Salle, Chicago, Ill. November 30-December 2, 1938. Dr. F. A. Imler, Secretary, Box 187, Kansas City, Kans.

United States Live Stock Sanitary Association. Hotel La Salle, Chicago, Ill. November 30-December 2, 1938. Dr. L. Enos Day, Secretary, 3933 Drexel Blvd., Chicago, Ill.

Nebraska State Veterinary Medical Association. Lincoln Hotel, Lincoln, Nebr. December 13-14, 1938. Dr. Jno. D. Sprague, Secretary, David City, Nebr.

South Dakota Veterinary Medical Association. Carpenter Hotel, Sioux Falls, S. D. December 15-16, 1938. Dr. G. E. Melody, Secretary, Gettysburg, S. D.

Immunological Studies on Equine Encephalomyelitis*

By ADOLPH EICHHORN *and* RALPH W. G. WYCKOFF, *Pearl River, N. Y.*

The rapid spread of encephalomyelitis through the horse population of western states has made the control of this virus disease a problem of immediate importance. Fortunately, extensive field and laboratory work over the last few years has supplied knowledge that can be utilized to provide adequate and widespread protection.

In the Americas, two forms of infectious equine encephalomyelitis are now recognized. The so-called eastern strain of the disease is known to occur only in the tidewater regions of the Atlantic seacoast. No cases of the western strain disease have as yet been reported east of the Appalachian Mountains, but it is prevalent in many states west of the Mississippi River, in western Canada and South America. Except for the greater severity of the eastern strain in horses and susceptible laboratory animals, the symptoms of the two forms are practically identical. The fundamental work of Meyer[1] and his collaborators, however, has shown that the causative viruses are antigenically unlike. Immunity to one virus gives no protection against attack by the other and, for all practical purposes, the two strains are to be considered as distinct diseases having similar clinical manifestations.

HYPERIMMUNE SERUM

It has repeatedly been shown that animals recovering from attacks of either eastern or western encephalomyelitis possess an effective immunity. Active immunization safely carried out is thus a satisfactory mode of protection. It has also been demonstrated that the serum of recovered animals, and particularly of hyperimmunized animals, contains antibodies capable of the *in vitro* neutralization of the virus. Such sera have been widely employed in the hope that they would provide passive immunity to exposed animals. It has been hard to evaluate the actual worth of these sera but at best they are too costly to make and they could convey too brief an immunity to be effective agents for the widespread control of the disease.

LIVE VIRUS VACCINE

There are two ways of actively immunizing horses against encephalomyelitis, depending upon whether a "live" or a "killed" vaccine is the agent. Immunization with "live" virus has been tried with both strains. Live virus vaccination has the advantage that, if it takes, an excellent immunity is set up. This procedure is, however, attended with danger with viruses like those of encephalomyelitis which are not readily modified. The dose must be very carefully controlled. If too much is given, the animal may manifest the disease, while if too little is given, it will be protected. Such control is both difficult and costly because dilute encephalomyelitis virus deteriorates rapidly on standing. Where complete eradication lies within the realm of possible attainment, as is true of this disease, live virus vaccination may also be undesirable because it may create an inexhaustible reservoir of virus by producing carriers.

KILLED VIRUS VACCINE

Effective "killed" virus vaccines evade these objections. We are indebted to Drs. Shahan and Giltner,[2] of the U. S. Bureau of Animal Industry, for showing that a formalinized vaccine made from diseased horse brains had protective action when injected into susceptible animals. This basic fact, since amply confirmed, is further supported by careful consideration of the field data of last year. An extensive study of the properties of this horse-brain vaccine has not been made because, while it undoubtedly gives a measure of protection to horses, it

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

is too weak to give a solid protection to susceptible laboratory animals. A more potent vaccine for guinea pigs can be prepared from diseased guinea-pig brains, and Cox and Olitzky[3] have carried out a thorough quantitative study of such a vaccine. They have demonstrated that their formalinized vaccine will protect guinea pigs against more than a thousand lethal doses of virus injected intracerebrally, that this immunity lasts for periods of several months at least and that the vaccine is a far more stable product than the active virus. They have also compared vaccines that were completely killed with ones retaining a demonstrable measure of active virus. It is of the greatest theoretical and practical importance that their best vaccines were ones in which no residual virus could be found.

HORSE-BRAIN VACCINE

As an instrument for the control and ultimate eradication of equine encephalomyelitis, horse-brain vaccines have two serious limitations. They are weak vaccines and hence must be used in large amounts to yield useful protection. The supply is strictly limited by the number of horses that one can afford to sacrifice for their manufacture. With these restrictions in mind, one of the present writers (Adolph Eichhorn) suggested to Dr. J. W. Beard, of Duke University, and another of the writers (R. W. G. Wyckoff) the desirability of an improved vaccine. They showed in studies[4] carried out partly at Duke University, partly at the Rockefeller Institute in Princeton and partly at the Lederle Laboratories that a formalinized vaccine made from diseased chicken embryos[5] is especially effective. In the following discussion we wish to describe some of the properties of this new vaccine which is one capable of imparting to horses a high degree of immunity and can be made in unlimited quantities.

CHICK-EMBRYO VACCINE

There are many reasons to believe that the immunizing capacity of a formalinized vaccine is a function of the virus concentration in the tissue used. On this basis, it is easy to understand why formalinized horse brains furnish a relatively weak vaccine. To form an estimate of the amount of virus in the brains of horses prostrate with the disease, we have made dilution titrations of the virus content of a number of brains as they were harvested for the production of the western horse-brain vaccine. Of twelve such brains, which represented all those taken within a given period of time, two were infectious at a dilution of one to 10,000 while disease could not be produced from two at dilutions greater than one to 100. Of the remainder, three could not be diluted more than one to 500, four had a dilution end-point of one to 1,000 and one was not infectious in 1 per cent suspension. From these results, we may say that the brain of the average horse dying of western encephalomyelitis has an end-point of about 10^{-3}. An egg embryo, diseased with western virus, has an average dilution end-point of 10^{-8}. Under favorable circumstances, it reaches 10^{-9}. In none of the large scale batches of virus that we have thus far handled for commercial vaccine production has the virus content been less than 10^{-7} infective doses per gram. The average fully diseased chick embryo thus has a virus concentration that is 100,000 times greater than that of the average horse brain. In other words, there is as much virus in a single embryo as in about 500 horses.

The superiority of the vaccine made from embryos is easily demonstrated by such an experiment as the following. Two groups of several guinea pigs each should be selected, one to be vaccinated with two 0.5-cc doses of chick vaccine at a week's interval, the other to receive two 1-cc doses of a standard horse-brain vaccine. Two weeks after the second injections, all the animals should be given from 100 to 1,000 lethal doses of virus directly into the brain (a 10^{-3} suspension of a well-diseased guinea-pig brain in 0.15-cc amount is a suitable inoculum). All of the horse-brain vaccinated pigs will die promptly with typical paralysis. None of the animals receiving chick vaccine should manifest the disease. We have made this experiment with horse-

brain vaccines of three different manufacturers with the same result each time. A typical protocol is shown in table I. The chick part of this experiment has been adopted as the routine test of the efficacy of each production batch of vaccine. These batches now exceed 60 in number and every one to be released has provided complete protection to all inoculated guinea pigs. Chick vaccines have already protected many hundreds of guinea pigs against such massive infections with eastern and with western strain viruses.

Experiments with horses, though naturally less extensive, have provided an equally clear cut demonstration of the superior protective action of the chick vaccines. We have now injected a dozen chick-vaccinated horses intracerebrally with doses of western virus which promptly brought disease and death to an equal number of unvaccinated controls. Every chick-vaccinated horse has remained perfectly healthy without manifesting any symptoms of disease. When small infecting doses of virus were used, most but not all horses receiving the old horse-brain vaccine were protected. The inferior degree of this protection was made evident by increasing the infecting dose. Thus in the experiment[6] of table II all three horse-brain-vaccinated horses died, even more promptly than the controls, while the chick-vaccinated horses have remained completely unaffected. We wish to emphasize the fact that in all these experiments with horses, the virus has been administered directly into the brain substance. This is, as far as we are aware, the first time that horses have been given a degree of immunity that would enable them to withstand this severe mode of infection.

TABLE I— *A comparison of the immunity in guinea pigs provided by chick and horse-brain vaccines.*

ANIMAL	OBSERVATIONS (DATE)									
	4–29	4–30	5–1	5–2	5–3	5–4	5–5	5–6	5–7	5–8
CHICK VACCINE										
A	A	A	A	A	A	A	A	A	A	A
B	A	A	A	A	A	A	A	A	A	A
C	A	A	A	A	A	A	A	A	A	A
D	A	A	A	A	A	A	A	A	A	A
E	A	A	A	A	A	A	A	A	A	A
F	A	A	A	A	A	A	A	A	A	A
HORSE-BRAIN VACCINE										
G	A	A	M	D	—	—	—	—	—	—
H	A	A	A	A	D	—	—	—	—	—
I	A	A	A	M	D	—	—	—	—	—
J	A	A	A	M	D	—	—	—	—	—
CONTROL ANIMALS										
1	A	A	M	D	—	—	—	—	—	—
2	A	A	A	M	M	D	—	—	—	—
3	A	A	A	M	M	D	—	—	—	—
4	A	A	M	D	—	—	—	—	—	—
5	A	A	A	M	D	—	—	—	—	—
6	A	A	M	D	—	—	—	—	—	—

*=Active.
†M=Moribund.
‡D=Dead.
§Virus dose: 100—1000 M.L.D.

QUESTIONS

Having at hand a vaccine capable of giving a solid immunity to treated animals makes possible a new approach to fundamental problems concerning the nature of the viruses of these diseases and the character of the immunity that can be established against them. We have been especially interested in seeking answers to the following questions: (1) How lasting is the immunity conveyed by vaccination with killed virus vaccine? (2) How rapidly does protection develop after vaccination has been begun? (3) What are the keeping qualities of a formalinized vaccine of this type? (4) How stable is the antigenic structure of the virus itself? In other words, are we today dealing with the same disease that prevailed several years ago?

DURATION OF IMMUNITY

To the question of the duration of the immunity conveyed by chick vaccination, we have as yet no answer but we have set up an experiment that will give us this answer by next season. Several groups of horses have been vaccinated with the chick vaccine. At intervals of three months, a group is receiving virus intracerebrally according to the scheme outlined in the preceding experiment. With the information that these experiments will furnish, we will be in a position to say whether vaccination is necessary every season.

The powerful immunity that is conveyed by chick vaccine makes it possible for the first time to determine whether two vaccinations are necessary and to follow in quantitative fashion the way in which immunity is established following administration of vaccine. We have found that most but not all guinea pigs are completely protected against massive intracerebral inoculation of virus one week after receiving a single dose of vaccine. In tests routinely made on production lots of vaccine, most guinea pigs were protected one week after a first vaccination. It is thus clear that a substantial degree of immunity develops quickly after chick vaccination but, since with these same vaccines we have never lost a pig receiving dual

TABLE II—*A comparison of the immunity in horses provided by chick and horse-brain vaccines.*

ANIMAL	TEMPERATURES (DATE)								
	6–17	6–18	6–19	6–20	6–21	6–22	6–23	6–24	RESULTS
CHICK VACCINE									
1	100.0	100.0	100.2	100.4	100.2	100.0	100.4	100.2	No symptoms
2	99.4	99.6	99.4	99.4	99.8	99.4	99.6	99.4	No symptoms
3	100.6	100.2	100.4	100.4	100.0	100.0	100.2	100.0	No symptoms
HORSE-BRAIN VACCINE									
4	100.4	100.0	100.8	100.2	102.6	103.4			Paralyzed, destroyed
5	100.0	99.8	100.4	99.6	103.0				Paralyzed, destroyed
6	99.6	100.0	100.6	103.0	104.6	101.4			Paralyzed, destroyed
CONTROL ANIMALS									
7	101.0	100.6	102.0	102.4	103.6	104.0			Paralyzed, destroyed
8	100.2	100.0	100.0	99.6	101.6	103.0	104.4		Paralyzed, destroyed
9	100.0	99.6	101.0	100.6	103.8	104.0			Paralyzed, destroyed

Dose of chick vaccine—10 cc.
Dose of horse-brain vaccine—25 cc.
Dates of vaccination: 5/3/38 and 5/17/38. Date of virus injection: 6/16/38.

vaccination, it is evident that protection is still mounting after a week. Such results emphasize the importance of vaccinating several weeks in advance of exposure for the optimum of protection. Nevertheless, their demonstration of the rapid development of measurable immunity after one vaccination is evidence that this product can be profitably used even under epidemic conditions.

Since beginning the study of this vaccine, we have learned much about its stability and keeping qualities. It is, in the first place, immeasurably more stable than the virus from which it is made. We do not yet know how long chick vaccine maintains unimpaired its immunizing capacity. We do know, however, that a vaccine made six months ago and stored meanwhile in a refrigerator is still giving all vaccinated guinea pigs complete protection. Chick vaccine against encephalomyelitis and probably all similar vaccines have lives which shorten rapidly as the temperature is raised. It cannot be left at a room temperature of about 70° F. for more than two or three weeks and in the incubator it is worthless after three days. Especially since these incubator temperatures do not exceed those commonly encountered in the field, we cannot emphasize too much the importance of a refrigerated handling of vaccine both during shipping and during storage before use.

IDENTITY OF VIRUSES

We have been especially interested in the question of whether or not the essential characteristics of encephalomyelitis viruses and the diseases they induce modify with time or through passage in different susceptible animals. For example, is the disease we had in 1933 the same as the one that is starting up again this year? Is the disease in Texas just like that in California or Canada, or is the disease after prolonged passage through mice, guinea pigs or chick embryos, let us say, a very different thing from that encountered in the field? We who are seeking to control this disease must have answers to these questions for two reasons. Obviously, we must know whether a vaccine made in the laboratory protects against virus as it occurs in the field, as well as against the virus with which it is made. Likewise, if live virus vaccination is under consideration, it is imperative to know whether either infectiousness or antigenicity is greatly altered by successive passages through a different host. To gain information on these two points, we[7] have determined the immunity provided by the chick vaccination of guinea pigs against western virus from as widely different sources as possible. We have employed vaccines made from a virus derived from the 1933 epidemic and meanwhile passed through guinea pigs.

Animals vaccinated with this vaccine have been inoculated according to the scheme outlined above with 100 to 1,000 lethal doses of the following strains of western virus: (1) Homologous western virus from the 1933 outbreak; (2) a strain of 1933 California virus which has been carried in mice without interruption for the last five years by Dr. Peter K. Olitzky, of the Rockefeller Institute; (3) homologous virus after 60 subsequent generations through egg embryos; (4) two of the U. S. Bureau of Animal Industry's strains of the 1937 disease, one from Texas, the other from Iowa. The vaccinated pigs have been completely protected against each of these viruses. This indicates for encephalomyelitis virus an antigenic stability that greatly simplifies control of the disease, for it permits us to make a vaccine from the virus from one locality with assurance that it will protect against disease as it occurs in another locality and another year.

SUMMARY

1. The chick embryo has proved a most suitable medium for the propagation of equine encephalomyelitis virus. When fully diseased the virus concentration within it is 100,000 times that in horse brain.

2. Formalinized chick-embryo vaccine is incomparably more effective for immunization than horse-brain vaccine.

3. With chick vaccine it is possible to provide complete protection to horses against intracerebral inoculation of many fatal doses of virus.

4. Chick vaccine possesses such a high degree of potency that it is feasible to standardize every batch for protective value prior to use.

5. With the aid of the chick vaccine, it is possible to inaugurate an effective control of equine encephalomyelitis.

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DISCUSSION

DR. H. E. CURRY: I was very much interested in the statement by Dr. Eichhorn to the effect that experimental work was being carried on in an effort to determine the changes that may take place in the virus. In Missouri, in 1935, we had only a few cases of equine encephalomyelitis. In 1936, no cases were reported. However, in 1937, the disease was widespread and approximately 4,900 animals were affected.

This year, in the so-called Delta section, in southeast Missouri, where the disease was not reported last year, we have had many cases reported. However, clinically, the animals do not show symptoms that have been described. I would like to know if Dr. Eichhorn can give us some information on this point.

DR. EICHHORN: From time to time, we have had reports from different parts of the country on the clinical manifestations of the disease, the variance of the clinical manifestations, and also the occurrence of the disease in seasons when equine encephalomyelitis was not supposed to occur. Very extensive tests have been made, if I recall correctly, in Montana, with brains of these so-called irregular cases, and also in Colorado. While the inoculation experiments have been negative, I do not believe that we are justified in assuming definitely that it has not been the typical virus disease of equine encephalomyelitis. We know very well that in the early investigations, in 1930, by Dr. Karl F. Meyer and his co-workers, they had the idea of first transmitting the disease to test animals, and, if you go back even to the old Kansas horse disease outbreak, in which I participated, we know very well that the man in the field was the best man we could find. We never succeeded in transmitting the disease. I believe, however, personally, that that was only because we did not carry out the work long enough, and with enough perseverance. Had we undertaken such work as they did in California, probably the virus nature of the disease would have been established many years before.

I believe that while the symptom complex is not very clear-cut in all cases, we nevertheless should consider that most of these cases really are induced by the virus.

DR. JOHN REICHEL: Mr. Chairman, I want to offer my congratulations to Dr. Eichhorn on his presentation. I do not think he left us with any doubt that this chick-embryo vaccine is a killed virus, but what makes him so certain that it is actually killed and that a dead virus immunizes?

DR. EICHHORN: We know very well that a certain school suggested that immunization with dead viruses cannot accomplish anything. We know also that various other people have stated that if such virus is used in sufficient quantity, then immunity will follow. As far as our test on immunity is concerned, we have not more evidence, possibly, than with rabies virus. We know that when we inoculate—whether it is horses or other test animals—with this thing that we call formalinized vaccine, we never succeed in producing the disease. We call it killed vaccine in rabies. Cold dead. It is. Therefore, I think that with just as much justification as the evidence gives us in rabies, we can call this a dead virus.

Two Drugs Held Illegal Under New Federal Act

Cinchophen and aminopyrine have been tagged by the U. S. Food and Drug Administration as dangerous drugs when they are promiscuously distributed.

One section of the newly enacted Food, Drug and Cosmetic Act holds a drug to be illegal if it is dangerous to health when used in the dosage, or with the frequency or duration, suggested in its labeling.

The Food and Drug Administration has warned manufacturers and dealers that the marketing of these two drugs in interstate commerce, under labeling "which may result in their use by the general public," will be regarded as violating this provision of the law. This does not restrict use of the drugs when prescribed by physicians.

Cinchophen has been widely used in the treatment of arthritis, but the toxic effects have proved so serious that most physicians now refuse to use it.

Aminopyrine is a powerful pain-killer but has an injurious effect on the blood and has been rejected by many physicians on this account. It has caused many deaths in the United States.

Field Application of Hog Cholera Tissue Vaccine*

By WILLIAM H. BOYNTON *and* GLADYS M. WOODS, *Division of Veterinary Science, University of California, Berkeley, Calif., and* F. W. WOOD, *Cutter Laboratories, Berkeley, Calif.*

The cost of hog cholera in this country for the year 1932, according to Kinsley,[1, 2] was about $500,000 per million hogs as compared with the low figure of $175 per million hogs in Canada. This stupendous difference in costs, which does not appear to have been reduced materially over the succeeding years, is sufficient evidence of the pressing need for improved methods of hog cholera control. Of the adjustments required for the control of this disease, the most immediate should be directed toward the substitution of an efficiently-functioning but less hazardous immunizing agent for the serum-virus method in use at the present time.

The purpose of this report is to show that, with the adoption of a proper procedure for handling Boynton's tissue vaccine,[3, 4] this product is an acceptable substitute for serum and virus, judged from (1) the degree of immunity developed, (2) the absence of severe reactions and disease-producing properties, and (3) the reduction of losses from hog cholera "breaks"[5] and concurrent infections[6] incident to the use of unmodified virus.

NATURE OF TISSUE VACCINE

Before discussing the field trials, it may be advisable to explain briefly what this vaccine is and to reiterate its advantages and limitations.

Tissue vaccine, of the type made at present, is a fine suspension of ground tissues from a pig which was inoculated with potent virus and then destroyed six or seven days later when the infection had reached its height. The virus in these tissues is treated with eucalyptol for a sufficient length of time, until it is so modified that it cannot produce cholera infection. We then test each lot for potency by vaccinating six pigs, leaving two controls in the same pen with this group of six, and later inoculating both vaccinated and control pigs with virus. A period of at least two weeks following the second injection is required in order to develop a satisfactory resistance to virus inoculation. The controls should die of cholera and the vaccinated group should be protected. Lots which are found to be of insufficient potency are immediately discarded. A number of highly potent lots are now customarily mixed together and the mixture is retested in the manner just described, before it is used in the field.

The efficiency of serum-virus as a means of developing immunity is undeniable, but this method is so attended with risks of "breaks" attributable to the virus itself and to flare-ups of secondary infections, such as necrotic enteritis and others, that it has fallen short of its goal. Tissue vaccine, on the other hand, having lost its disease-producing properties as a result of treatment with eucalyptol, does not cause a systemic reaction in the pig and cannot, therefore, bring about a virus "break." In direct contrast to the frequent fatal results following the serum-virus immunization of pigs afflicted with necrotic enteritis, pneumonia, verminous infestations, or other pathological conditions, the results with tissue vaccine indicate its far greater safety.

Inasmuch, however, as a pig, suffering from one or another of these infections, would not withstand serum-virus treatment, a question arises, based on laboratory observations, whether immunity could be developed in this animal, by the tissue-vaccine method or any other method, which would be sufficient to protect it against severe exposure to hog cholera, or whether, under adverse conditions, immunity which

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

would be adequate in normal animals, would be overcome by the recurrence of one of these latent infections. Whatever the answer to this question may be, the pig, at least, does have a chance of surviving the intercurrent infection, when tissue vaccine is used.

A further advantage is that tissue vaccine has been shown by field and laboratory experiments to be incapable of transmitting cholera to any unvaccinated pigs which may be present in the herd. Also, the normal feeding ration may be maintained during the period of immunization without any ill effect upon the animal.

Tissue vaccine is limited in that it has no curative properties whatever. It cannot take the place of antiserum and is not intended to do so. Tissue vaccine is of value as a preventive only, and one that does not carry with it the possibility of establishing new centers of infection.

The advantages described, obviously outweighing the limitations of tissue vaccine, should be evidence enough that this method is manifestly safer than serum-virus treatment. That it is definitely capable of inducing an adequate immunity against cholera in healthy pigs is clearly demonstrated in the field trials.

FIELD TRIALS

The field trials, which are summarized in table I, were begun in late March, 1936, and thus far a total of 15,125 pigs have been vaccinated. This total comprises 30 herds in widely scattered areas over 14 counties of California. With the exception of one garbage-fed herd, these are all grain-fed animals, maintained under average farm conditions. The subsequently described procedures applied to the herds were adapted to the conditions encountered in each herd.

Herd 1, the first selected for trial, was one in which losses, chiefly from necrotic enteritis, following serum-virus administration, ranged from 25 to 60 per cent over a period of years. In addition, infestations of *Ascaris* were common. The earlier trials with tissue vaccine were carried out on sucklings; that is, the first injection of 5 cc of vaccine was given when the pigs were six weeks old, and the second injection at eight weeks of age, their weaning time. For a year now, the procedure has been to vaccinate the healthy pigs shortly after weaning them and follow with a second injection two or four weeks later.

Unthrifty pigs, or those sick from conditions other than cholera, when presented for first treatment, are vaccinated, segregated, and fed upon a special diet. The survivors among them are revaccinated at a later date. The plan of vaccinating pigs after weaning has been instituted, in this herd and those treated later, because observation, both in the laboratory and in the field, has shown that weaned pigs develop a greater degree of immunity than sucklings. The use of tissue vaccine at this time includes, also, one yearly injection of the breeding stock until the duration of the immunity has been definitely established under field conditions.

In the period of 27 months which has elapsed since the use of tissue vaccine was begun on herd 1, 8,188 pigs have been vaccinated with the result that not one field case of hog cholera has occurred, and losses from enteritis have been reduced to about 6 per cent, a fact which gives the owner the expectation of 700 more pigs this year than were produced from the same number of sows in previous years of severe loss incident to serum-virus administration.

HANDLING AN INFECTED HERD

The mode of handling an infected group is illustrated by herd 4, in which hog cholera, necrotic enteritis, and pneumonia were existent. To control the active cholera infection present in this herd and forestall any "breaks" for which the tissue vaccine might be condemned as responsible, all pigs of weaning age were treated with anti-hog cholera serum before vaccination was begun. When, after a lapse of three weeks, the beneficial effects of the antiserum had worn off and no further signs of hog cholera infection were observed, these pigs were given the first injection of vaccine. As a result of meeting the situation in this manner, no cases of hog cholera have been reported among the 1,069 pigs vaccinated over a period of twelve months. In addi-

tion, the incidence of enteritis and pneumonia has been considerably diminished.

Another example of a herd occupying infected premises is herd 3, in which hog cholera losses were continuous. In the latest outbreak, this disease was introduced through a group of infected feeders brought to the premises, and, although serum-virus was administered, fatalities persisted. In this instance, although hog cholera was present in the herd, use of the tissue vaccine method was begun. The survivors in the feeder group and the exposed and sick animals in the other groups were given a large dose of serum. The unexposed weanlings were injected with tissue vaccine, isolated in a pasture, and provided with a caretaker. Among the next group vaccinated, hog cholera was found present in one litter and all of the pigs in this litter were given a large dose of serum. The other litters were apparently healthy and were segregated in a pasture, vaccinated, and weaned. Two of the sick litter died. Three weeks later, the survivors were vaccinated. The feeders which had received serum alone also were given tissue vaccine three weeks later. In a total of 731 pigs vaccinated over a period of 14 months, further losses from hog cholera have not occurred and secondary complications have abated.

Most of the other herds included in these field trials either had current infections or histories of previous outbreaks similar to those in the herds previously discussed. Indeed, one owner of a small group had lost all but three after serum-virus treatment; in another instance, only one sow remained. Besides the post-vaccination "breaks" which resulted in about 40 per cent mortality among the younger stock of a third group, several of the sows, previously immunized to hog cholera by the simultaneous method, succumbed to this disease. Other herds were pure-bred stock and the owners were unwilling to subject their animals to the risk of the simultaneous method. In short, the owners of all the herds feared the presence of hog cholera virus on their premises.

Vaccination was conducted on all these herds according to the conditions encountered. If no infection was present, injections of vaccine were given at once; if infection was found, serum treatment was administered. Of the two herds of feeders, herd 7 had been on the premises for some time before the owner requested tissue vaccine, and since no infection had appeared, vaccination was immediate. The other herd (10) was held for about two weeks before vaccine injection, in order to await development of any infection which might be wrongly attributed to the vaccine, if the "break" occurred before immunity could be produced.

Since it will be noted from table I that no incidence of hog cholera has been reported on the premises occupied by any of the vaccinated herds and a lowering of mortality from secondary conditions has been brought about through the use of tissue vaccine, the results are clearly as gratifying in all the herds as in those given particular mention.

INTERCURRENT INFECTIONS

In earlier trials on the garbage-fed herd, some confusion arose as to the results because of the presence of other infections in the group. Of the last group of 70 pigs vaccinated, 16 which received two injections, one before and one after weaning, and nine which were given three injections, one before and two after weaning, were inoculated with virus and left in contact with the entire vaccinated group. Two of the 16 pigs given one treatment before weaning and one after weaning died. All of the remaining virus-treated and contact pigs survived. The group of 95 pigs most recently vaccinated received three injections, one before and two after weaning, and so far seven mortalities have been reported, three due to pneumonia, and four to intestinal disturbances.

In spite of these reassuring results, these trials cannot yet be considered other than experimental. Since it has been customary to treat this herd with serum-virus at five weeks of age, and again when the pigs are weaned at twelve weeks, in order to develop a satisfactory immunity, it is possible that, until the weaning method is changed, three doses of tissue vaccine may be required, one

TABLE I—*Field trials with hog cholera tissue vaccine. Total number of pigs vaccinated to June 10, 1938—15,125.*

HERD	HISTORY OF HERD AT TIME OF VACCINATING	PIGS VACCINATED	CLASS AND AGE	RESULTS
1	Post-vaccination losses, due chiefly to necrotic enteritis and *Ascaris* infestation, ranged from about 25 to 60 per cent	8,188	Sucklings, weanlings and breeders	No hog cholera during the past 27 months; reduction in losses from necrotic enteritis to about 6 per cent
2	Previous hog cholera "breaks," pneumonia, and *Ascaris* infestation	1,177	Weanlings	No cases of hog cholera; few cases of hog flu
3	Current hog cholera	731	Weanlings and imported feeders	No cases of hog cholera in vaccinated pigs
4	Hog cholera, pneumonia and necrotic enteritis	1,069	Weanlings	No cases of hog cholera; marked reduction in losses from pneumonia and enteritis
5	Current pneumonia and necrotic enteritis; previous post-vaccination losses	585	Weanlings	No cases of hog cholera; marked reduction in losses from pneumonia and enteritis
6	Current pneumonia	290	Weanlings	No cases of hog cholera; reduction in losses from pneumonia
7	No infection	310	Imported feeders	No cases of hog cholera reported
8	Some unthriftiness present; no hog cholera	148	Weanlings	No cases of hog cholera reported
9	Previous severe hog cholera "breaks"; no current infection	148	Weanlings and breeders	No cases of hog cholera reported
10	No infection	209	Imported feeders and weanlings	No cases of hog cholera reported
11	No infection	69	Weanlings	No cases of hog cholera reported
12	Previous severe post-vaccination "breaks"; no current infection	56	Weanlings	No cases of hog cholera reported
13	No infection	178	Weanlings (purebred)	No cases of hog cholera reported
14	No infection	17	Weanlings (purebred)	No cases of hog cholera reported
15	No infection	17	Weanlings (purebred)	No cases of hog cholera reported
16	No infection	163	Weanlings (purebred)	No cases of hog cholera reported
17	No infection	19	Weanlings (purebred)	No cases of hog cholera reported
18	Previous post-vaccination "breaks"; no current infection	115	Weanlings	No cases of hog cholera reported
19	Severe post-vaccination "breaks" from cholera and necrotic enteritis	169	Weanlings	No cases of hog cholera; marked reduction in necrotic enteritis
20	Nucleus of a purebred herd; no infection	10	Breeders	No cases of hog cholera reported
21	Previous losses from necrotic enteritis	185	Weanlings, feeders and breeders	No cases of hog cholera; marked reduction in necrotic enteritis
22	No infection	278	Weanlings	No cases of hog cholera reported
23	No infection	22	Weanlings	No cases of hog cholera reported
24	No infection	19	Weanlings (purebred)	No cases of hog cholera reported
25	No infection	89	Weanlings and breeders (purebred)	No cases of hog cholera reported

TABLE I—*Field trials with hog cholera tissue vaccine. Total number of pigs vaccinated to June 10, 1938—15,125.*—(Continued)

HERD	HISTORY OF HERD AT TIME OF VACCINATING	PIGS VACCINATED	CLASS AND AGE	RESULTS
26	Previous severe hog cholera "breaks"; current pneumonia and enteritis	144	Weanlings and feeders	No cases of hog cholera reported; reduction in losses from pneumonia and enteritis
27	Current pneumonia and *Ascaris* infestation	245	Weanlings	No cases of hog cholera reported; reduction in losses from pneumonia
28	No infection	27	Weanlings and breeders	No cases of hog cholera reported
29	Previous severe hog cholera "breaks," necrotic enteritis, and *Ascaris* infestation	112	Weanlings	No cases of hog cholera reported; reduction in losses from enteritis
30	Current infection common to garbage-fed pigs	336	Sucklings	Trials still regarded as experimental; very encouraging results

at five weeks, and two after weaning, to achieve adequate protection.

PROCEDURE ADOPTED

Haphazard methods of vaccination cannot be depended upon to produce suitable results with tissue vaccine any more than such methods are reliable in immunization against any other disease. Competent handling is essential. From the field experiences related in this report, an outline of procedure has been devised which embraces several important provisions.

First: If the premises are free from cholera, vaccination may be undertaken immediately. Two injections of vaccine are necessary; an interval of at least two weeks must elapse between the injections, although a longer period may be permitted to suit the convenience of the attending veterinarian. Previously immunized breeding stock should receive one injection yearly.

The dosage is 5 cc at each injection and the site of injection is the armpit, to which a good disinfectant should be applied before the injection is made. In this work, tincture of iodine has been used. Observation of such aseptic precautions has prevented entirely the formation of abscesses which may follow injections into undisinfected areas. Aseptic precautions are necessary in the handling of instruments and other equipment required in the process of vaccination; clothing and equipment which have been used in serum-virus immunization should not be used for tissue vaccine administration for fear that some virus may have remained on them and may affect the pigs before the tissue vaccine can induce a proper resistance to the disease.

Second: Exposed pigs in herds which harbor cholera infection or into whose midst infection is introduced by pigs brought in from other places or located on premises in which virus is present, must be treated with antiserum before vaccination can be begun. If, after three weeks, when the temporary immunity conferred by the antiserum has disappeared, no further signs of infection are present, the pigs are ready to be immunized.

Third: Due to the possibility of unknown exposure, feeders should be held for observation for at least two weeks before being vaccinated. If they are introduced to augment an already existing herd, they should be kept separate from that herd for this period of time. These measures are urged not from fear of the effect of such exposure on the already immunized animals in the herd, but rather to guard against the vaccination of exposed or infected pigs, which obviously would be to no purpose, to prevent the contamination of clean premises by such pigs, and to safeguard

pigs of pre-vaccination age in the existing herd. In order to provide sufficient protection for feeders which are to be shipped, vaccination should be so arranged that a period of at least two weeks is allowed between the time of the second injection and the time of shipping.

The problem of the so-called chronic cases, which are in reality chronic infections, makes it incumbent upon the veterinarian to become sanitarian as well. While the absence of unmodified virus will, after a moderate length of time, bring about an elimination of cholera from a herd, secondary complications may persist to some extent, particularly if they are existent in the breeding stock. This situation resolves itself into a problem of sanitation which the experienced veterinarian can meet. In fact, knowing how and when to vaccinate requires the training of a veterinarian and, for these reasons, tissue vaccine is not to be entrusted to the layman.

In summarizing briefly the results of field trials with tissue vaccine, the diminution of losses from secondary complications clearly indicates the safety of this method, in respect to post-vaccination "breaks," and a completely negative record of hog cholera cases subsequent to the use of tissue vaccine on various premises occupied by 30 herds, totaling 15,125 pigs, is unmistakable evidence of the progress which can be and is being made in the control of hog cholera.

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DISCUSSION

QUESTION: How long does it take to establish immunity with this method of treatment?

DR. BOYNTON: About three weeks. We prefer giving two injections, since we wish to apply this vaccine in the safest possible manner.

DR. G. H. ROBERTS: What is the dose?

DR. BOYNTON: The dose is 5 cc. It is much easier to immunize a 50- or 60-pound pig, or an 80- or 100-pound pig, than it is a recently weaned pig. The recently weaned pig is growing to such an extent that his tissues are changing, and his immunity does not hold so well as it does in an older, more mature pig. That is why we prefer giving two injections—to reinforce the immunity. If we would give more injections in immunizing animals against diseases such as cholera, a stronger immunity would be established.

DR. F. P. CAUGHMAN: How much does it cost, compared with serum and virus?

DR. BOYNTON: I do not know. The Cutter Laboratories are to produce this vaccine commercially and they will set the price.

QUESTION: I would like to know how soon after weaning you make your first injection.

DR. BOYNTON: Just as soon as the pigs are weaned or within two weeks afterwards. One thing to do is to get them away from their mothers and on their own.

QUESTION: Would you advise using three injections on garbage-fed hogs?

DR. BOYNTON: Until the procedure for garbage-fed hogs is more firmly established, yes. We recommend one injection before weaning and two after weaning.

QUESTION: Is this vaccine on the market at the present time?

DR. BOYNTON: No, it is not.

Associated Serum Producers Launch New Radio Program

A new fall and early winter series of radio broadcasts, dealing with seasonal live stock health problems, is being launched over a large group of radio stations this month by the Associated Serum Producers under the sponsorship of the American Foundation for Animal Health. The broadcasts are being made in connection with farm programs and market report periods on leading stations in farm areas. All the broadcasts urge the farmer to call his veterinarian at the first sign of sickness in his live stock and to rely on veterinary advice for accurate diagnosis and treatment.

Among the subjects being covered in the broadcasts are rickets, hog cholera, garget, fall losses in feeders, grass poisoning, cornstalk disease, and similar health problems. The radio material follows the same plan which has worked out so successfully in the broadcasts which have been put on by the Associated Serum Producers in previous campaigns.

Some Newer Drugs Used in Veterinary Medicine*

By D. W. ASHCRAFT, *Columbus, Ohio*
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When we speak of "some newer drugs used in veterinary medicine," we are not thinking so much of when they were discovered, but, rather, when they were used clinically. The mentioning of newer drugs immediately brings to mind sulfanilamide, prontosil, metrazol, mandelic acid, nicotinic acid, benzedrine sulfate, acriflavine dyes, lentin, rotenone, Lang's solution and pontocaine. In the short time allotted to this paper it would be impossible to deal properly with all these drugs. Therefore, I shall discuss sulfanilamide and prontosil more or less in detail and make a few remarks about metrazol.

SULFANILAMIDE

Since sulfanilamide therapy is becoming so important in the treatment of human infections, it is only natural to desire to explore the scope of its use in veterinary medicine. An attempt will be made to review the progress of research on sulfanilamide and prontosil, particularly those papers dealing more directly upon animal therapy.

Schulte,[1] reviewing the history of sulfanilamide, states that sulfanilamide did not appear suddenly but was the product of numerous investigations which extended over a period of 30 years. In 1908, Gelmo,[2] who was interested in the azo dyes for the dye industry, was the first to mention sulfanilamide. Succeeding years brought forth more compounds until 1917-19, when Jacobs and Heidelberger[3, 4] stated that many of these substances were bactericidal *in vitro*. In 1935, Domagk,[5] director of the experimental pathological laboratory of Elberfeld, Germany, pointed out the therapeutic possibilities of sulfanilamide and used the drug in a few clinical cases. Since 1935, a number of new compounds have been synthesized, but at a symposium held at Johns Hopkins Hospital, last April, it was generally accepted that these compounds were not superior and in some cases were inferior to sulfanilamide.

One physician remarked recently that the therapeutic use of sulfanilamide bids fair to be as great a boon to the human as was insulin. Long and Bliss[6] have shown that sulfanilamide is effective for all Beta hemolytic streptococcal infections. A number of clinical reports [7-12] indicate that sulfanilamide is also of value in the treatment of meningococcal, gonococcal, Welch bacillus and melitensis infections and certain urinary tract infections due to staphylococci, *E. coli, B. aerogenes, B. pyocyaneus,* group B Beta hemolytic streptococci and *B. proteus.*

So far, little research work has been carried out to determine the efficacy of sulfanilamide upon the infectious organisms found in diseases of animals. Johnson and Miller[13] and Klein and co-workers[14] have made an excellent start in the right direction in determining the value of sulfanilamide in mastitis of cattle. A great deal of research is needed on all animals to determine dosage, physiological action, toxicity, and the organisms that are inhibited or destroyed.

PROPERTIES AND PREPARATIONS

Sulfanilamide appears as white crystals, 0.8 per cent soluble in cold water. It is administered orally, but may be used subcutaneously, where oral administration is impossible. One hundred cc of sterile physiologic sodium chloride solution is heated in a flask to the boiling point. The flask is then removed from the flame and the contents of a 1-gram ampule or 3 five-grain tablets of sulfanilamide are added. This solution is ready for parenteral injection

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

as soon as it is cooled to body temperature. If it is allowed to cool appreciably below body temperature, the drug will begin to crystallize out of solution. Sulfanilamide may be secured in tablet or powder form. Tablets are of 5, 7.5, 15 and 30 grains, in bottles of 25, 100, 500 and 1,000. The drug is also available in 1-gram ampules, in boxes of 5 and 25 ampules, and in powder form in bottles containing 500 grams.

Prontosil is a red dye, 4 per cent soluble in cold water. It is marketed in 5 per cent solution in bottles of 125 cc. It may be given intravenously or subcutaneously and is used primarily in human medicine when sulfanilamide is not tolerated. However, Long[15] believes in the superiority of sulfanilamide to the extent that he advises that as soon as practical, one should return to oral administration of sulfanilamide. Prontosil upon analysis yields 11 grains of sulfanilamide per 100 cc of solution.

DOSAGE

The suggested initial daily dose of sulfanilamide for the human is 1.0 gram per 20 pounds (9.1 kg) of body weight, in six divided doses at four-hour intervals. Long stresses the importance of maintaining an even and regular schedule of therapy, in order that an adequate blood-level of free sulfanilamide may be maintained. In severe cases he advocates a 10 to 20 mg per cent blood level of free sulfanilamide, while in mild cases he feels that a 4 to 7 mg per cent is satisfactory. In clinical cases in the human, that could not tolerate sulfanilamide, Long administered 3.4 cc of prontosil per kg subcutaneously every four hours.

Marshall, Cutting and Emerson[16] administered 0.1 and 0.2 gm per kg of sulfanilamide on numerous occasions to many dogs without any observable symptoms. On this basis, about six grams should be easily tolerated by a 20-lb. dog, if given in six divided doses at four-hour intervals. Others have recommended a somewhat smaller dose, such as 1 grain per pound of body weight, not exceeding 60 to 75 grains daily, in divided doses.

One cc of prontosil per kg may be administered subcutaneously every four hours to a dog, if sulfanilamide is not tolerated.

One of the first clinical cases of mastitis in cattle, in which sulfanilamide was used, was reported by Stevens.[17] He administered 75 grains twice daily for four doses, or a total of 600 grains in four days.

Allott[18] administered sulfanilamide in three cases of mastitis, giving 30 gm four times daily. He reported a marked improvement in these cases.

Johnson and Miller[13] treated six cows with sulfanilamide, giving 75 grains twice daily for three days, a total of 450 grains. In the second part of the experiment four animals were given one-half ounce, twice daily for eight days, a total of 3,600 grains. The drug was sprinkled on the feed in the dry state. There were no unfavorable effects, except a slight diarrhea. Milk production remained normal. Klein and coworkers[14] found one cow that did not tolerate a dosage of 25 gm four times daily for five days. When the animal stopped eating grain, the drug was discontinued. Two days following, when the dose was dropped to 20 gm three times daily, the temperature rose to 103° F. Appetite failed and milk production decreased. Osgood[19] has suggested that a concentration of 1 mg of sulfanilamide in 100 cc of blood may bring about desired results, basing his conclusions on experiments *in vitro*. Following this suggestion, Klein[14] then gave smaller doses, which maintained a concentration of 1 to 2 mg of sulfanilamide in the blood and milk. He recommends the following dosage for cattle: a large single dose (30 to 45 gm) followed in twelve hours by 15 gm, and then doses of 7.5 to 15 gm every twelve hours for the course of the treatment.

The dosage may be summarized as follows:

Sulfanilamide:

Large animals (horses and cattle): From 20 to 45 grams given in six divided doses, orally. Small animals: one grain per pound of body weight, not exceeding 60 to 75 grains, in six divided doses, orally.

Prontosil:

Large animals (horses and cattle): A daily dose of 100 to 150 cc of a 5 per cent solution,

given in four to six divided doses, subcutaneously or intramuscularly.

Small animals: A daily dose of 1 cc per kg of body weight in four to six divided doses, subcutaneously or intramuscularly.

Recommendations have been made, for both sulfanilamide and prontosil, to give a large initial dose the first day, and then gradually decrease the dosage on the second and third days, maintaining the size of the dosage of the third day throughout the course of the treatment. This procedure is followed in order to reach a high concentration of the drug in the tissues rapidly and to maintain this level as constant as possible.

ABSORPTION AND ELIMINATION

Marshall and co-workers[20] have reported on the absorption and excretion of sulfanilamide. Four hours after oral administration, fecal analysis showed that in normal dogs 99 per cent of the sulfanilamide is absorbed from the intestinal tract. In one case in which a dog was heavily infested with worms, only 75 per cent was absorbed. The free form of sulfanilamide is present in the tissues just as soon by oral administration as it is by parenteral injection. In fact, Long[15] reports that with the parenteral route there is more marked variation in the blood levels of the drug, as determined at various intervals of time, and because of this he prefers oral administration.

The concentration of sulfanilamide in various body fluids (saliva, pancreatic juice, spinal fluid and pleural effusions) appears to be substantially equivalent to the concentration in the blood. Klein[14] reports that peak concentration occurs in the blood-stream in eight to twelve hours and lasts for 36 to 48 hours. In some cases peak concentration occurred in the milk at the same time as in the blood, and in other cases earlier. Methods of determining the concentration of sulfanilamide in the blood, urine and milk are reported by Marshall,[20] Klein,[14] and others.

Nearly 100 per cent may be recovered from the urine when equilibrium between intake and output is established. It takes from two to three days to establish this equilibrium and the same time to free the body of the drug after it is discontinued. Sulfanilamide is excreted more slowly when the patient has impaired renal function.

In the dog sulfanilamide appears to be excreted unchanged in the urine, while in the rabbit, large amounts are excreted in the conjugated form (acetyl sulfanilamide), which is inactive. It is possible that some of the domestic animals may evidence this phenomenon. If so, it will necessitate larger doses of the drug in urinary infections, since an adequate concentration of free sulfanilamide must be maintained in the urine.

Since sulfanilamide and prontosil are excreted almost entirely by the kidneys, Long[15] advocates limiting the human patient to 3,500 cc of water a day, in order to keep the concentration of the drug at the proper level. Therefore, in treating urinary infections in animals, we should limit the daily consumption of water. These drugs cause an increased excretion of Na and K and a fall in CO_2 combining power, producing cases of acidosis. This condition can be avoided by giving one-half as much $NaHCO_3$ as you give of sulfanilamide at each dose, whether it be sulfanilamide per os or prontosil by parenteral administration.

TOXICITY AND INCOMPATIBILITIES

Apparently animals show less toxicity to these drugs than do humans. It is highly possible that when the proper blood concentration is maintained for a time, toxic symptoms and individual idiosyncrasies may be evidenced. Halpern and Mayer[21] determined that the toxic dose for dogs is 1 gram per kg. Dogs given this dose show ataxia, athetotic movement followed by rigidity, tonic-clonic convulsions and, finally, coma. Marshall, Cutting and Emerson[16] gave dogs 1 gram per kg, causing the animals to show salivation, vomiting, diarrhea, hyperpnea, excitement, muscular weakness, ataxia, signs of stimulation, and depression of the central nervous system, leading to a condition of spastic rigidity of the limbs and hypesthesia. All symptoms

disappeared in ten to twelve hours and the animals appeared normal.

Klein[14] demonstrated toxicity in one cow by administering 25 gm three times daily for five days and, two days following, giving 20-gm doses. Toxic symptoms were produced, lack of appetite, rise in temperature and lessened milk production.

It is not known whether other toxic symptoms shown by the human, such as cyanosis, methemoglobinemia, allergy, agranulocytosis, urticaria, and acute hemolytic anemia, occur in animals. Long[15] regards acute hemolytic anemia in the human as one of the most serious in the course of sulfanilamide therapy. The cause is unknown, but it apparently is an example of individual idiosyncrasy. Blood transfusions are indicated.

No saline cathartics should be administered with sulfanilamide therapy, especially magnesium sulfate, since there is some evidence that these drugs facilitate the formation of sulfhemoglobin. This greenish-brown compound is formed when hydrogen sulfide combines with hemoglobin. Sulfhemoglobin is formed in the blood-stream during life, especially when the gas occurs in very large amounts.

When toxic symptoms appear, give plenty of fluids. Never give laxatives.

Long[15] observes that alcohol and sulfanilamide tend to complement each other in their toxic effect. Therefore, it would appear foolhardy to supplement sulfanilamide therapy with alcohol. While treating 345 patients at Johns Hopkins Hospital, Long[15] has supplemented sulfanilamide therapy with acetosalicylic acid, phenobarbital, iron compounds, digitalis and arsphenamine.

THERAPEUTICS

Osgood[19] suggests that therapy be instituted at the earliest possible moment, since the effectiveness is greater if the number of organisms present is not too great.

A number of cases of acute tonsilitis in dogs, showing the presence of streptococcic infection, have been treated at the Veterinary Clinic at the Ohio State University. These cases have responded quickly with sulfanilamide treatment.

A rather unfavorable case of purulent metritis in a five-year-old dog appeared at the clinic last month. She had a history of two cesarean sections and one case of dystokia. Her physical condition was so poor that it was deemed advisable to give full doses of prontosil. A remarkable recovery was recorded in four days.

In certain cases of urinary infection, sulfanilamide therapy may be indicated. Long[15] views with alarm the widespread and uncontrolled use of this drug in the treatment of mild infections of the urinary tract. It is his belief that sulfanilamide should be used in urinary infections only after conservative methods of treatment have failed.

Allott[18] observed marked improvement in three cases of chronic mastitis in cows. Other investigators, including Johnson and Miller[13] and Udall,[22] reported little or no effect in cases of chronic mastitis of cattle. Udall[22] makes an interesting observation concerning the use of sulfanilamide in cases of acute mastitis. He says:

> In many of these cases, the acute symptoms subside without any treatment. Naturally, if a drug is given, it would receive credit in bringing about improvement.

Klein[14] reports slight improvement in similar cases. More recently, Ramsay[23] reports the successful use of full doses of sulfanilamide in the treatment of two cases of acute mastitis.

Clinical reports upon the use of sulfanilamide in cases of equine purpura hemorrhagica, metritis in cattle, fistula of the withers in the horse, influenza of the horse, metritis in the brood mare, navel-ill, canine distemper, and otitis media in dogs and swine, have been favorable. Especially favorable reports have been received from Udall[22] in cases of calf pneumonia. He believes the mortality rate has been materially reduced by sulfanilamide therapy.

SUMMARY AND CONCLUSIONS

Sulfanilamide offers a chemical aid to the practice of veterinary medicine. An observation made by Merillat,[24] in the fall of 1937, still holds true. He stated:

> The list of diseases of animals for which sulfanilamide may be scientifically employed remains to be made up. * * * * There is still much to be learned about the dosage.

While we have made some progress, there is much research yet to be done, to determine which infective organisms in animals are susceptible, the dosage, colorimetric determinations of the blood, urine and milk concentration, free and conjugated form of the drug in urine, and the toxicity. Long's[15] researches indicate that we should make daily hemoglobin and total white-cell counts.

Clinical reports are decidedly helpful in furthering our knowledge. We should make careful clinical observations, take temperatures frequently, keep animals quiet and make daily hemoglobin determinations and total white-cell counts. With these data we can make rapid strides, which will determine the efficacy of the drug and teach when and when not to use it.

METRAZOL

Without a doubt, many small-animal practitioners have had the unfortunate experience of having an animal stop breathing under surgical anesthesia. At such a time, one desires one of the best respiratory stimulants. Barlow,[25] basing his judgment on more than 1,600 separate experiments, rates analeptics by the degree of improvement in respiration, circulation, and reflex excitability; degree of shortening the usual stages of recovery; and the margin of safety of effective dosages of each agent. Naming them in order, from high to low, they are as follows: picrotoxin, metrazol, ephedrine, artificial respiration, "coramine," "icoral," strychnine, and caffeine sodio-benzoate. His reports have been corroborated by Jackson[26] and Henderson and Sparks.[27]

On the other hand, Camp[28] concluded from his experimentations on rabbits that his results do not seem to warrant the use of metrazol therapeutically. He reports a dilation of the capillaries in the abdominal organs and a failure in many cases to stimulate the respiratory center. Morris and Eastman[29] have found this drug to be of value in practice in shortening the duration of an anesthetic and as a stimulant to the circulation and respiration in pneumonia cases, in conjunction with dextrose therapy. They make it a routine practice to include ampules of metrazol with their instruments, sutures and other surgical equipment.

EXPERIMENTATION

In our laboratory experiments, we have found metrazol to be one of the best respiratory stimulants for dogs. The respiratory effects of this drug exceeded all other preparations as to speed and degree of action. Excessive dosage of metrazol produced clonic convulsions similar to excessive doses of coramine or picrotoxin.

We purposely injected large doses of morphine sulfate intravenously, producing paralysis of the respiratory center. In a few minutes respiration ceased, the heart action remaining good. In nine out of ten cases, the injection of 1 cc of metrazol intravenously caused the dog to respire normally in one to three minutes. No other analeptic produced this speed and degree of action.

DOSAGE

We have found the 1-cc ampule to be the most economical, since in many cases 0.5 to 1 cc is sufficient to bring about recovery. Each 1-cc ampule represents 1.5 grains of the drug. Metrazol may be administered intravenously, intramuscularly or subcutaneously. If necessary, the drug may be repeated in a few minutes.

ABSORPTION AND ELIMINATION

The absorption is rapid, especially when administered intravenously. Elimination occurs in the urine in from one to three hours.

SUMMARY

From our experience, metrazol appears to be one of the quickest and most powerful of the respiratory stimulants. It should be of great assistance in those cases where the threshold of the respiratory center is raised to such a high degree by anesthetics that the rate and amplitude of respiration are diminished to a dangerous level.

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DISCUSSION

DR. R. W. JACKSON: Were these toxicity experiments run on normal animals or animals suffering from some condition? Were the toxicity experiments run just to see how much they could stand?

DR. ASHCRAFT: All these toxicity experiments reported were carried out on normal animals, mice, rats, rabbits and dogs. Despite the widespread use of sulfanilamide, a limited amount of investigation has been done on toxicity. Most workers conclude that the toxicity of sulfanilamide for these animals appears to be relatively small, but the substance is not devoid of toxicity.

These toxicity experiments were run to see what symptoms these large doses would produce and, in those that recovered, to observe the after-effects.

DR. JACKSON: In the case of a horse you might be treating, what symptoms would you expect to see in case of an overdose?

DR. ASHCRAFT: If we may draw conclusions from a very limited number of cases of toxicity experiments on horses, we would say that danger of poisoning is small. Probable symptoms that one would observe are: temperature rise, salivation, diarrhea, lack of appetite and trembling.

Sportsmen Protest Rabies Quarantine

On the grounds that hunting dogs rarely spread rabies and, therefore, they should be exempt from quarantine, 2,000 members of the Detroit Sportsmen's Congress have appealed to the Commissioner of Agriculture for a revision of the dog quarantine, now effective in eleven southern Michigan counties.

Speed Record for Dogs

Record speed for a dog is 36 miles per hour, set by a greyhound over a quarter-mile course. *Hunting and Fishing* points out that most bird dogs average about 20 miles per hour, but are able to keep going irregularly for two or three hours and undoubtedly could outdistance the greyhound in a long race.

Changes in the Blood Cellular Elements in Relation to Diagnosis and Prognosis *

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The progress of both human and veterinary medicine, during the last decade, can be attributed largely to the advances made in the science rather than the further development of the art. The former has unlimited possibilities, whereas, the latter has limitations which are insurmountable.

The demand for more accurate diagnosis can be met only by a more intelligent interpretation of not only subjective and objective symptoms, but in addition the unveiling of the obscure or occult symptoms by means of special clinical and laboratory tests.

In the diagnosis and control of communicable disease, the veterinary profession has been and still is in the front. As regards the non-communicable and non-infectious diseases, we are falling behind.

Time will not permit me to discuss the use of various laboratory tests which may be useful in diagnosis and prognosis, so I will confine my remarks to those things which offer the most promise of being helpful to the veterinarian.

VALUABLE EXHIBIT

For more details regarding these tests and for specific information, I wish to call to your attention the exhibit of "Clinical Laboratory Methods in Small-Animal Practice." This exhibit by Drs. Allison, Green and Morris will give you a very good idea of the progress being made along this line and I would advise every one of you not only to visit it but to study the material presented.

Hematological studies in human medicine are today practically indispensable in the diagnosis of many diseases. Much of the valuable knowledge obtained from these tests has been the result of long usage, which has been correlated with clinical and necropsy findings together with considerable experimental work. The fundamentals of these tests are the same for both animals and man; however, their significance and clinical value will differ with each species. For physiological reasons one test may be of considerable value in one species and not in another, even though the disease may be the same in both. Even in the same species there may be differences which influence the value of certain tests. We cannot stop here, as we still have to consider the breed, sex, age and other factors.

In order to apply any laboratory test properly to clinical studies of any given species, it is necessary to consider the following: (a) the physiology of the organ or organs involved, (b) the pathology of the disease processes affecting such organs, and (c) the range of normality for the test with regards to species, breed, age and sex.

FUNCTIONAL TESTS

It should be kept in mind that most of the laboratory tests, with the exception of some bacteriological tests, are not in themselves diagnostic, but merely elicit additional symptoms which can not be detected otherwise. The great majority of them are really functional tests, that is, they determine whether an organ or group of organs are functioning properly.

There may be hypofunction, or hyperfunction of an organ; however, the type or degree of abnormality may be secondary to dysfunction of another organ, *e. g.*, in man a low basal metabolic rate indicates hypothyroidism.

The value of clinical laboratory tests is not only to aid in diagnosis, but they are

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

often useful as a guide for the treatment after the diagnosis is made. In addition, laboratory tests are often a great help in prognosis.

VALUE OF A BLOOD-COUNT

Let us discuss the value of a single blood-count, which is a laboratory test with which you are all familiar and which is now used by some veterinarians, especially those in small-animal practice. By this procedure the presence or absence of anemia is accurately determined. In addition, it tells you the exact degree of anemia present. By correlating the red cell-count with the hemoglobin the type of anemia is revealed.

If oxalated blood is collected for the count, then this can be centrifuged and the volume of cells in relation to whole blood can be ascertained, and then by calculating the mean corpuscular volume, the exact size of the red cell is known. In some cases of anemia the cells are smaller than normal (microcytic) and in others they are larger than normal (macrocytic). Microcytic anemia may be secondary to many diseases, whereas, macrocytic anemia is due to liver dysfunction, improper diet (especially lack of meat), or to disease of the stomach. The treatment in such cases would consist in the feeding of liver, or injection of liver extract. In some cases the simple restoration of meat to the diet may suffice. How often this type of anemia occurs in animals I am unable to say, but it should be looked for especially in those cases where there has been an attempt to make a vegetarian out of a carnivorous animal, also in those cases which show liver or gastric symptoms.

A low hemoglobin in relation to the number of red cells may be due to iron deficiency, in which case the administration of iron is indicated.

The total count of the leukocytes in itself is of limited value. It is, therefore, advisable to make a differential count in all cases. The number of each type of cell present then can be determined. This is readily done by using the figures obtained in the total and differential counts.

DIFFERENTIAL COUNTS

An increase in the number of neutrophils (neutrophilia) is common in acute infections, especially those due to cocci, loss of blood, infarcation, and leukemia.

A decrease in the neutrophils (neutropenia) is commonly seen in subacute and chronic infections and convalescence from severe acute infections, virus diseases and in cases of drug idiosyncrasy.

In chronic infections and during convalescence from acute infections the lymphocytes may be increased.

An increase in monocytes indicates tissue destruction.

Eosinophilia is common in parasitic diseases and allergic conditions.

The advisability of blood transfusions depends upon the state of the blood as determined by the cell-count.

In addition to the number of cells, a careful study for evidence of degeneration and immaturity should be made. This can be done at the same time the differential count is made. Particular attention should be paid to the maturity of the neutrophils. This is based upon the number of lobes in the nucleus. A few immatures are normally present. In infection there is a relative increase of the immature forms. This study of the neutrophils was first described by Arneth, in 1904.

In 1911, Shilling suggested a classification which has become very popular and useful. Several other classifications have been advocated and one of these may be mentioned because of its simplicity, namely, the filament-non-filament count of Farley *et al.*

When an increase of immature forms occurs in relation to the mature forms, it is called a "shift to the left." On the other hand, if there is an increase or greater lobulation of the cells, it is called a "shift to the right." The "shift to the left" is important and of clinical significance.

Degenerative changes in the neutrophils, such as toxic granulation, may be of significance in some animals. In some species the normal granularity of the neutrophils interferes with this observation.

So far we have discussed but a single blood-count and this has told us where we stand, but a subsequent count the next day, or later, will help a great deal in telling us where we are going. In anemia, subsequent tests will reveal improvement or the contrary. In infections a decrease of neutrophils with a diminished "shift to the left" is encouraging and the opposite is unfavorable.

OTHER BLOOD STUDIES

This gives us an idea of the information obtained from one simple blood study. Other blood studies, such as the determination of urea, creatinin, sugar, cholesterol and calcium, and the carbon dioxide-combining power of the blood, may in some cases prove helpful in diagnosis, prognosis or treatment.

Urinalysis has proved of value not only in the diagnosis of renal disease, but also in the detection of liver dysfunction, acidosis, and other conditions. In small-animal hospitals, this laboratory procedure should be routine.

Fecal examinations are routinely done by veterinarians for the detection of parasites.

In the not far distant future there may be developed a practical method for lumbar puncture. This would aid in the diagnosis of diseases involving the central nervous system. Practical methods for liver and kidney function tests may prove of value.

WHO WILL PAY THE BILL?

The question probably will be asked, "Who will pay for all these tests?" In answering this question I should remind you that no one animal will have to have all these tests. If you are equipped to perform any of these tests or have them done, and have the knowledge that they are of value to the patient, then I think you will find many among your clientele who are willing to pay the extra fee, if they think it necessary.

Of course there are poor animals, the same as poor humans, and the problem is the same in both cases. If your fee is sufficient, you can certainly do some of these tests, in those cases which are of particular interest, for nothing.

When conducting tests be sure of your technic and keep accurate records so that your findings can be compiled and reported. The more reports of clinical laboratory tests, the more progress will be made. It is just as important to eliminate valueless tests as it is to establish the use of valuable ones.

The federal and state governments have given and are still giving generous financial support for laboratory work in connection with the communicable diseases. In some instances the eradication of disease is dependent upon the adaptation and use of laboratory tests, *e.g.*, in glanders, dourine, rabies, and other lesions. The reason for this support is obvious. These diseases affect either the health, pocketbook or food supply of mankind. The development and adaptation of laboratory tests for the non-communicable diseases of animals is of the greatest benefit to the animals themselves. Therefore, it has lacked financial support from man. It is my sincere belief that, if the societies interested in the welfare of animals would give financial support to investigations regarding the adaptations of clinical laboratory tests to animals, for the benefit of the animals themselves, it would be greatly appreciated by the whole animal kingdom.

DISCUSSION

DR. A. R. THEOBALD: I would like to ask Dr. Boerner one question. I don't know how much work he does on small animals; I know his work is largely confined to humans. I am wondering if he knows that in many of the young dogs brought to the veterinarian for immunization against a virus disease, such as distemper, a low percentage of hemoglobin is frequently found, usually 60 to 70 per cent. Would this be considered an unhealthy sign for immunization, even though the total white count might be very good?

DR. BOERNER: That is very questionable. It brings out a very important point. I am unable to answer it. Probably Dr. Theobald has a better answer than I have. It does bring up a very important point, and that is the establishing of normals for the different ages, as well as different sexes.

We know, for instance, that a baby or an infant, at time of birth, has a high hemoglobin which rapidly goes down the first few months of life. It goes down far below the adult's normal range, so in children the average may run

about 10 grams of hemoglobin per 100 cc of blood, which would be very low for an adult, but as the child grows it increases, so at 15 or 20 years of age he carries hemoglobin anywhere from 12 to 18 grams.

And so I believe when you speak of young animals, especially puppies, that we have to learn more about what the normal is for the various ages, and then we will be in a better position to answer your question. What we really need is not an average normal for these different breeds or ages, but we need to know the range of 90 per cent of the normals, how low and how high they are, so that a report falling within that range can be considered normal. Without this knowledge we do not know how high to go to be normal, or how low.

We must establish better normals and more normals, and one of the biggest problems in the field of veterinary medicine is that of the multiplicity of species. Each species is a separate problem, and you can not take any of these tests and apply them to any species of animal, because it may not be of value in that animal. The normals given for humans are not necessarily adapted to animals.

Dedication of Abbott Laboratories New Research Building

On October 7, at North Chicago, Ill., the new Research Building of the Abbott Laboratories was dedicated with appropriate ceremonies. The new building contains laboratories where every facility is provided for conducting research work along bacteriological, chemical and pharmaceutical lines. There is also a micro-analytical laboratory. The library has a capacity of 20,000 volumes and an air-conditioned auditorium provides seating accommodations for 800 persons.

The morning was spent on a trip of inspection through the plant, including not only the Research Building but the various manufacturing departments. Luncheon was served in the cafeteria at the plant. At 2 o'clock, the guests assembled in the auditorium for the afternoon program. Dr. Ernest H. Volweiler, Director of Research, presided. Following a welcoming address by Mr. S. DeWitt Clough, president of Abbott Laboratories, three prominent speakers addressed the group of research workers who came from all parts of the country to attend the dedication. The speakers and their subjects were as follows:

"The University and the Public Welfare," Dr. Karl T. Compton, President, Massachusetts Institute of Technology.

"The Spirit and Task of Research," Dr. Herbert M. Evans, Professor of Anatomy and Director of Experimental Biology, University of California.

"Research and Public Health," Dr. Thomas Parran, Surgeon General, United States Public Health Service.

A banquet was served at the Palmer House, in Chicago, in the evening. Dr. Roger Adams, head of the Department of Chemistry, University of Illinois, presided. He first introduced Mr. E. H. Ravenscroft, chairman of the Board of Abbott Laboratories, who greeted the assembled guests. He was followed by the following speakers:

"The Contributions of Organized Chemistry," Dr. Harrison E. Howe, Editor, *Industrial and Engineering Chemistry*.

"The Scientific Development of Drug Standards," Dr. George D. Beal, Assistant Director, Mellon Institute.

"The Contributions of Medicine to Public Welfare," Dr. Morris Fishbein, Editor, *Journal* of the American Medical Association.

At table 7, Dr. E. E. Sweebe, in charge of the Veterinary Department of Abbott Laboratories, had as his guests: Dr. Ward Giltner, Dean, Division of Veterinary Science, Michigan State College, East Lansing, Mich.; Dr. N. S. Mayo, Chairman, A. V. M. A. Committee on Education, Highland Park, Ill.; Dr. Charles Murray, Dean, Division of Veterinary Medicine, Iowa State College, Ames, Iowa; Dr. J. V. Lacroix, editor of the *North American Veterinarian*, Evanston, Ill.; Dr. E. C. Khuen, North Shore Animal Hospital, Evanston, Ill.; Dr. D. M. Campbell, editor of *Veterinary Medicine*, Chicago, Ill., and Dr. H. Preston Hoskins, Secretary-Editor, American Veterinary Medical Association, Chicago, Ill.

Veterinarians Help Observe National Dog Week

In observance of National Dog Week (September 18-24), the Scott County (Iowa) Humane Society sponsored a free "dog clinic" in Davenport. Lectures on the care of dogs were given by various members of the Society. Throughout the week, two Davenport veterinarians, Drs. J. E. Kellberg and O. T. Douglass, conducted free examinations and rendered first-aid treatment.

Nutrition as a Factor in the Incidence of Fowl Leukosis*

By W. J. BUTLER, D. M. WARREN *and* H. L. HAMMERSLAND
Montana Livestock Sanitary Board, Helena, Mont.

Considerable work has been done on the etiology, pathology and classification of fowl leukosis and its associated manifestations. Little information, however, is available at the present time relative to the control or the prevention of this disease. Unfortunately, in many instances the clinician has been forced, for lack of better information, simply to advise the poultryman whose flock is affected with this disease to dispose of that flock, thoroughly clean and disinfect the premises and buildings, and start all over with newly purchased birds.

From the standpoint of a sanitary official responsible for the control of infectious-contagious diseases and from the standpoint of a clinician, to advise the poultryman to destroy his entire flock is not productive of added prestige and, in our experience, as far as fowl leukosis is concerned, is not always sound advice. It is from the standpoint of a clinician and a control officer, therefore, that we have attempted to outline some corrective measures.

Fowl leukosis is so widely spread and is causing such tremendous losses to the poultry industry that corrective measures must be made available. Otherwise, the industry will be faced with economic annihilation.

There has been a great deal of confusion and variance in the terminology which pertains to the general subject of fowl leukosis. We recognize the various manifestations and classifications used relative to this disease. However, we have combined these various leukemic and paralytic conditions for the purpose of this study under the general term of "fowl leukosis."

*Presented at the seventh-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

INFLUENCE OF DIET ON DISEASE

It has been our observation, extending over a good many years, under range as well as farm conditions, that various diseases of animals and fowls are many times directly influenced by a proper or improper nutritional balance. We have observed that some so-called infectious-contagious diseases apparently may be prevented from becoming active in the animal body or may disappear following corrective changes in diet.

We are of the opinion that all so-called virus diseases are not caused by a living entity. Some viruses may be complex chemical compounds which become activated through a chemotactic impulse. From a physical standpoint there is a tremendous difference between the animate and the inanimate. However, from a chemical standpoint there may be very little difference. It is not at all impossible that these activating agents may be carried from place to place, such as on the shoes and clothing of attendants, and in this way stimulate actual living organisms.

In the past few years, due to the excessive drouth prevailing in certain sections, we have observed that, in all classes of live stock, due to the temporary impotency of our natural feeds, we have been forced, in order to maintain a proper nutritional balance, to supply, through the addition of vitamin and mineral concentrates, the factors that climatic conditions have diminished in our feeds. It must also be realized that in the domestication of poultry certain unnatural practices have been resorted to, such as selective breeding, artificial housing, forced maturity and artificial feeding.

These practices have resulted in more than doubling natural production and to compensate for this increased production

we have not given sufficient consideration to a proper nutritional intake. In many cases poultry producers have reduced the intake of green feed due to a commercial necessity and to the clamor of the general public for light-colored egg yolks. These practices have resulted in a weakening of the physical structure of the breeding stock and a marked reduction in the natural resistance of many basic flocks.

NON-PARASITIC ENTERITIS

It has been our experience that, in a great number of birds presented to us for postmortem examination, there is present a chronic enteritis, due to some factor which is not explainable on the basis of a parasitic invasion. Much reliance has been placed on commercial feeds which contain added marine oils to furnish some of the essential food elements. We found in our investigations that enteritis was generally associated with fowl leukosis and with the feeding of commercial feeds.

Fig. 1. Bird 186. Paralyzed in right leg. Received, May 6, 1938.

Our work also demonstrated that a considerable percentage of these feeds contain rancid marine oil. We realize that rancidity is highly conducive to enteritis, which interferes with assimilation, and that rancid oil is destructive to some vitamins. Therefore, it was with the thought in mind of reducing the incidence of enteritis and increasing the normal assimilation of foods that we endeavored to approach this problem from a clinician's viewpoint.

Realizing, of course, that there is considerable evidence to support the infectious theory and, also, the inheritance factor in leukosis, we feel that, while these theories are undoubtedly tenable, there is still the factor of natural resistance to consider and that perhaps the resistance factor in leukosis may be analogous to the resistance obtained by the proper balance of vitamin A in respiratory infections.

ENTERITIS REDUCED BY DIETARY CHANGES

In our first work, in an attempt to reduce the mortality in leukosis, we attempted to eliminate, as far as possible, any probability of rancidity in the marine oil by adding daily to our base ration fresh cod-liver oil containing 400 vitamin D units and 3,000 vitamin A units per gram. Also, to increase the general resistance of the flock, we provided fresh green feed daily. This practice resulted in a marked reduction in the enteritis and improved the general tone of the flock. However, it did not materially reduce the incidence of fowl leukosis, as evidenced by both the clinical symptoms and blood-picture.

We remembered the work of Adamstone[1] on the deliberate destruction of vitamin E and the production of a leukosis-like condition. We were also mindful of the work of Maynard,[2] in which he referred to vitamin destruction in the presence of rancid oils. We also had in mind the anti-oxidant properties of wheat-germ oil and vitamin E and realized that they would be a factor in stabilizing the vitamin content of the marine oils. With these factors in mind, we thought it advisable to follow a feeding procedure in which we combined fresh marine oil with wheat-germ oil in building up the resistance of flocks in an attempt to reduce the mortality due to leukosis.

It must be understood that all of our work has been done in various sections of Montana, under field conditions on the producer's own premises, as we do not have

facilities for an experiment station type of work.

FLOCK A

Flock A consisted of some 2,000 year-old White Leghorn hens. Since 1932, the owner has experienced increasing losses from fowl leukosis. He had used every effort to comply with sanitary recommendations. However, irrespective of strict compliance with a definite sanitary procedure with each new group of hens that was put into production, the excessive mortality continued.

In May, 1937, the owner requested that we suggest some type of control. Our investigation showed that the birds in this flock were pale and thin; some had gray eyes, some had diarrhea, and there was a general unhealthy appearance. The egg yolks were very light-colored. These birds were continuously housed and had no access to any green feed. Both postmortem and clinical examination showed evidence of fowl leukosis.

Fig. 2. Bird 186. Became normal in July, 1938, and shortly later developed bumble-foot, due to an injury. (Control pen.)

We selected a house of 150 birds and gave them a standard base ration free from marine oil, to which we added 1 per cent cod-liver oil daily. They were also allowed access to freshly cut alfalfa. Following this change in diet, there was a marked improvement in the general tone of the birds, an increase in the color of the egg yolk and a slight reduction in mortality as compared to the other 1,800 birds whose feed had not been changed.

This procedure was carried on for approximately 45 days. Noting the tendency toward improvement, we placed the entire flock on the experimental ration. We were not satisfied, however, with the results obtained and made a further change in the diet. We added to the base ration 0.5 per cent cod-liver oil and 0.2 per cent of cold-pressed wheat-germ oil daily. The feeding of fresh-cut alfalfa was continued. The entire flock was placed on this ration on July 14, 1937. In a short time following the last change in diet, a marked improvement was noted in the general appearance of the flock. Egg production was increased and there was a marked decrease in the flock mortality, as shown in table I.

TABLE I—*Flock mortality, November, 1935, to January, 1936, as taken from the owner's poultry-record book.*

MONTH	YEAR	BIRDS ON HAND	LOSSES
November	1935	1,875	26
November	1936	1,928	71
December	1935	1,849	18
December	1936	1,857	82
January	1936	1,831	54
January	1937	1,775	89
February	1936	1,777	44
February	1937	1,686	64
March	1936	1,733	41
March	1937	1,622	88
April	1936	1,692	54
April	1937	1,543	77
May	1936	1,403	47
May	1937	1,457	81
June	1936	1,356	40
June	1937	1,376	84
July	1936	1,316	79
July	1937	1,352	21
August	1936	1,257	99
August	1937	1,324	10
September	1936	1,683	84
September	1937	1,284	4
*October			
November	1937	1,280	7
December	1937	1,271	9
January	1938	1,262	8

*Records for the month of October are not available.

In July, 1937, 25 clinical cases of leukosis were taken from flock A. Segregation of similarly affected birds from this flock without their being subjected to treatment had been done prior to this with approximately a 100 per cent mortality. The 25 clinical cases referred to were segregated

from the flock and given 1 cc of wheat-germ oil intramuscularly every other day until three doses had been given. They were also given a ration containing 1 per cent freshly added cod-liver oil and 1 cc of wheat-germ oil per bird daily. On November 1, 1937, 18 of these birds, from a clinical standpoint, had made a complete recovery, were in good flesh and in production. Five of the birds had died, all of

Fig. 3. Bird 187. Paralyzed in both legs. Received, May 6, 1938.

which upon postmortem showed lesions of leukosis. Two of the birds which had a distinct iritis showed no improvement in the eye condition, but had gained materially in their general condition. However, these were destroyed November 1.

There was a progressive improvement in the physical condition of flock A throughout the fall of 1937 and the winter of 1937-1938 and they were profitable producers. In February, 1938, all but 200 birds of this flock were sold in order to make room for the chicks purchased at that time and designated later in this paper as flock D.

FLOCK B

Flock B consisted of 165 White Leghorn hens. These birds were placed in the laying-house on October 15, 1937. They started dying at the rate of two or three birds a week shortly after being placed in the laying-house. Postmortem examination showed lesions of leukosis. A differential blood-smear was made from each bird in the flock on November 26, 1937. Of the remaining 150 birds at this time, 36 showed a blood-picture indicative of leukosis.

Of this group of 36 birds, an intensive blood study was made on 25 birds, eleven of the group having died before work was started. These 25 birds were divided into two groups.

Each bird in group 1, consisting of eleven birds, all of which showed clinical symptoms as well as a blood-picture of leukosis, were given 2 cc of wheat-germ oil

Fig. 4. Bird 187. Became normal, June 28, 1938. Photograph taken September 27, 1938. (Control pen.)

intramuscularly the first day of treatment, followed by 1 cc intramuscularly every other day until each bird received a total of 7 cc. Each bird also received 3 minims of wheat-germ oil and 4 minims of cod-liver oil in the feed daily. Their protein ration was slightly decreased and their grain ration increased. Twenty-seven complete blood counts were made on these eleven birds, as shown in table II.

These birds are still living and in good health. Their average egg production is greater than the average production of the rest of the flock. After the final bleeding on May 24, 1938, they were allowed to run

outside and mingle with the rest of the flock for the summer.

Group 2 consisted of 14 birds. They were given one intramuscular injection only of 2 cc of wheat-germ oil. They also received 3 minims of wheat-germ oil and 4 minims of cod-liver oil daily in their feed. There were 113 complete blood-counts made on the 14 birds in group 2. The first blood-count, made in December, 1937, and the final blood-count, made in April, 1938, of the group 2 birds are shown in table III.

The remaining 114 birds in flock B received 3 minims of wheat-germ oil and 4 minims of cod-liver oil on their feed daily. From December 16, 1937, to June 15, 1938, nine birds in this flock died of leukosis.

TABLE II—*Blood-counts on eleven birds (group 1, flock B).*

BIRD	DATE BLED	HEMOGLOBIN (%)	LEUKOCYTES	ERYTHROCYTES	LYMPHOCYTES (%)	NEUTROPHILES (%)	EOSINOPHILES (%)	BASOPHILES (%)	DIFFERENTIAL BLOOD-SMEAR
76	12-16-37	85	23,110	2,840,000	79	15	0	6	Myeloblasts
	1- 6-38	85	23,550	2,980,000	78	18	4	0	Normal blood-picture
	5-24-38	95	20,660	3,000,000	72	24	3	1	Normal blood-picture
96	12-16-37	95	30,550	3,360,000	55	38	2	5	Basophilic myelocytes
	1- 6-38	95	21,770	3,210,000	73	25	1	1	Normal blood-picture
	5-24-38	100	21,110	3,330,000	70	26	2	2	Normal blood-picture
143	12-16-37	70	34,220	2,100,000	65	30	1	4	Myeloblasts
	1- 6-38	95	21,330	3,000,000	83	10	4	3	Budding of the lymphocytes
	5-24-38	100	19,880	3,100,000	76	22	1	1	Normal blood-picture
145	12-16-37	95	21,550	3,050,000	98	2	9	0	Lymphoblasts. Budding of the lymphocytes
	1- 6-38	95	21,550	3,110,000	85	10	4	1	New lymphoblasts
	5-24-38	95	21,550	2,980,000	70	24	3	3	Normal blood-picture
108*	12-16-37	80	26,330	2,650,000	61	36	0	3	Myeloblasts and anochromasia
112	12-16-37	95	38,220	3,270,000	85	0	13	2	Lymphoblasts and eosinophilic myelocytes
	1- 6-38	95	22,220	3,200,000	71	24	2	3	Slight budding of the lymphocytes
	5-24-38	95	21,110	2,980,000	73	22	3	2	Normal blood-picture
83	12-16-37	55	21,330	2,310,000	91	6	1	2	Lymphoblasts
	1- 6-38	80	21,110	3,100,000	90	7	1	2	Lymphoblasts and polychromasia
	5-24-38	90	20,660	2,960,000	72	25	2	1	Normal blood-picture
149†	12-16-37	55	50,440	2,220,000	86	12	1	1	Lymphoblasts, myeloblasts and anochromasia
118	12-16-37	60	29,110	2,620,000	77	20	1	2	Myeloblasts and anochromasia
	1- 6-38	90	20,660	3,080,000	79	17	1	3	Occasional myeloblasts
	5-24-38	90	21,770	2,970,000	75	23	1	1	Normal blood-picture
78	12-16-37	80	30,220	1,800,000	67	30	0	3	Megaloblasts. Slight budding of the lymphocytes
	1- 6-38	95	21,110	3,000,000	69	27	2	2	Budding of the lymphocytes
	5-24-38	95	19,550	3,110,000	68	30	1	1	Normal blood-picture
28‡	12-16-37	55	23,110	2,370,000	79	15	4	2	Normoblasts, lymphoblasts and anochromasia

*Leg-band lost; bird escaped into normal pen.
†Died 12-27-37. Diagnosis: leukosis.
‡Died 12-19-37. Diagnosis: leukosis.

TABLE III—*Blood-counts on 14 birds (group 2, flock B).*

BIRD	DATE BLED	HEMOGLOBIN (%)	LEUKOCYTES	ERYTHROCYTES	LYMPHOCYTES (%)	NEUTROPHILES (%)	EOSINOPHILES (%)	BASOPHILES (%)	DIFFERENTIAL BLOOD-SMEAR
91	12-20-37	75	22,220	2,230,000	92	6	1	1	Budding of the lymphocytes. Few lymphoblasts
	4-26-38	95	22,550	2,990,000	80	17	2	1	Normal blood-picture
95	12-20-37	65	20,000	2,100,000	68	20	12	0	Myeloblasts, anisocytosis anochromasia, polychromasia
	4-26-38	95	19,770	3,000,000	85	12	2	1	Normal blood-picture
40	12-20-37	80	21,660	2,500,000	64	34	2	0	Few myeloblasts
	4-26-38	100	21,110	3,110,000	78	28	0	0	Normal blood-picture
54	12-20-37	75	24,440	2,720,000	76	18	1	5	Anochromasia, polychromasia
	4-26-38	95	22,660	2,920,000	78	17	3	2	Normal blood-picture
47	12-20-37	75	20,880	2,780,000	79	18	1	2	Budding of the lymphocytes. Polychromasia
	4-26-38	75	20,110	1,990,000	89	8	1	2	Polychromasia, lymphoblasts
12	12-20-37	90	22,220	3,000,000	74	25	0	1	Few myeloblasts
	4-26-38	95	20,880	2,960,000	80	18	1	1	Normal blood-picture
122	12-20-37	90	29,330	3,330,000	81	16	1	2	Basophilic granules in the cytoplasm of the lymphocytes. Budding of lymphocytes
	4-26-38	100	22,220	3,510,000	75	23	1	1	Normal blood-picture
121	12-20-37	80	23,110	2,800,000	69	25	1	5	Polychromasia and budding of the lymphocytes
	4-26-38	95	21,330	3,110,000	76	20	2	2	Normal blood-picture
55	12-20-37	75	27,550	2,860,000	68	27	2	3	Neutrophiles, myelocytes, basophilic granules in the cytoplasm of the lymphocytes
	4-26-38	95	22,220	3,310,000	80	18	1	1	Normal blood-picture
57	12-20-37	80	22,220	2,580,000	68	27	2	3	Lymphoblasts. Budding of the lymphocytes
	4-26-38	95	21,110	3,000,000	76	21	2	1	Normal blood-picture
48	12-20-37	85	26,660	2,690,000	88	8	3	1	Few lymphoblasts. Polychromasia
	4-26-38	95	22,000	3,000,000	78	21	1	0	Normal blood-picture
49	12-20-37	75	33,330	2,800,000	88	10	2	0	Marked polychromasia, anochromasia, bi-nucleated lymphocytes
	2-21-38	95	20,660	2,960,000	70	28	1	1	Normal blood-picture
58	12-20-37	80	22,220	2,450,000	81	15	2	2	Few lymphoblasts. Polychromasia
	2-21-38	95	24,440	2,960,000	78	20	1	1	Normal blood-picture
126	12-28-37	80	26,880	3,010,000	60	35	3	2	Megaloblasts. Eosinophilic myelocytes
	2- 3-38	65	18,330	2,430,000	Impossible to count				Polychromasia. Eosinophilic and basophilic myelocytes. Megaloblasts

The balance of the flock improved in flesh, physical appearance and in production.

FLOCK C

Flock C consisted of 104 Rhode Island Red birds. These were purchased as sexed chicks on May 5, 1937. The owner of this flock is exceptionally well versed in poultry husbandry. We have had occasion to visit these premises many times in the past few years and there was no evidence or history of fowl leukosis on this ranch until October of 1937. These chicks were imported

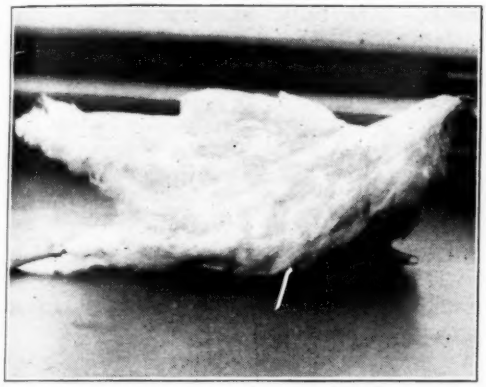

Fig. 5. Bird 191. Paralyzed in both legs. Received, May 6, 1938.

into Montana and came from the same hatchery as the birds in flocks A and D.

On October 1, 1937, 98 birds were placed in the laying-house. Between October 1 and February 5, 1938, 32 died. Postmortem examination of these birds showed that approximately all of them were affected with fowl leukosis. On February 5, differential blood-smears were made and counted on the remaining 66 birds. Thirteen of the 66 birds showed a blood-picture of leukosis. On February 14, all birds in the flock were given 2 cc of wheat-germ oil intramuscularly, 1 cc on February 16 and 1 cc on February 18. Starting February 14, there were added to the base diet 4 minims of cod-liver oil and 4 minims of wheat-germ oil per bird daily.

On April 12, 61 birds were bled and a differential blood-smear made and counted. During the interim between the bleedings on February 5 and April 12, one bird was destroyed on account of frozen feet and four died of leukosis. In the differential counts made on April 12, 1938, ten of the 13 birds that showed a positive blood-picture of leukosis on February 5 were normal. The three remaining of the 13 birds showed a blood-picture indicative of leukosis. Four birds that had a normal blood-picture on February 5 showed a blood-picture somewhat indicative of leukosis when reëxamined on April 12. Records were kept on this flock until May 15, 1938, when these birds were turned out and no further records were available.

FLOCK D

Flock D consists of 804 White Leghorn chicks. They were shipped to the owner of the flock listed as flock A and placed on

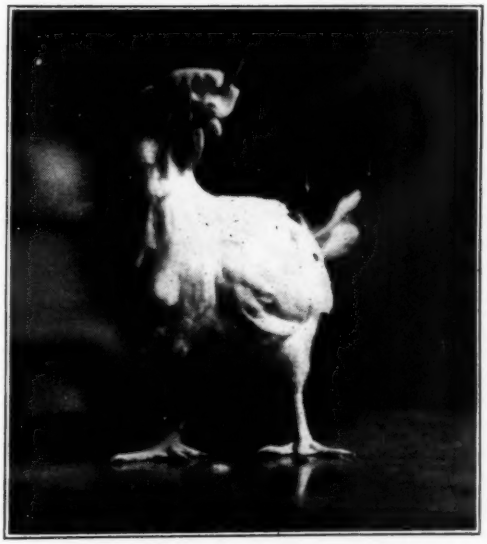

Fig. 6. Bird 191. Became normal, July 5, 1938. Photograph taken, September 27, 1938. (Pen 2, W. G. O.)

his premises on February 18, 1938. This is an experimental project, in which a definite agreement has been entered into with the owner of the ranch premises. This project is being financed in part by the Montana Livestock Sanitary Board.

These baby chicks were purchased from the same hatchery as the one which this owner had previously patronized and from which the birds in flocks A and C had been purchased. These chicks were brooded in battery brooders under the best possible

sanitary condition. At the end of five weeks, they were placed in houses which had been cleaned and disinfected under our personal supervision, and are being maintained entirely separate and apart from the birds that still remain on the ranch from flock A, the only contact being that the feeding is done by the same attendants. At the time these chicks were brought to the ranch, they were divided into seven groups and toe-marked for identification.

Each group is being fed on a different ration. Up to June 15, 30 cases of fowl leukosis have developed in flock D. There have been, however, four cases in our control birds to one case in those birds being fed what we consider a more balanced ration.

We trust to make a detailed report of this project at a later date, when the experiment has been terminated and when it may be possible to arrive at definite conclusions.

The four flocks mentioned in this paper are characteristic and picked from many flocks which we have had under observation. We feel that we are justified in stating that increased production and decreased mortality have followed corrective nutritional treatment in flocks affected with fowl leukosis.

SUMMARY OF OUR OBSERVATIONS

Increased production and a decrease in the incidence of fowl leukosis have followed changes in nutrition, which include the addition of medicinal amounts of cod-liver oil and cold-pressed wheat-germ oil.

The blood-picture of fowl leukosis is apparently constant.

The history of flock C supports the theory that fowl leukosis is transmitted through the egg.

The history of flock D indicates that fowl leukosis may be transmitted through the egg or possibly carried by mechanical means from non-clinical (carrier) cases to susceptible birds, and that direct contact is not essential for the development of fowl leukosis.

The history of flock D indicates the advisability of attempting to control fowl leukosis by merely destroying the flock, cleaning up the premises and making baby chick replacements.

In certain flocks it is indicated to reduce protein intake and increase the grain ration.

Where cod-liver oil and wheat-germ oil are used, the wheat-germ oil should be mixed with the cod-liver oil and added to the feed just previous to its use.

In the majority of our flock cases, we added 0.5 per cent cod-liver oil and 0.2 per cent cold-pressed wheat-germ oil to the base feed just previous to its use.

Marine oils added to feeds may become rancid. Rancidity impairs food values. Rancid oil may cause enteritis, which results in reduced resistance to disease and reduced production.

Large doses of cod-liver oil or wheat-germ oil may be harmful and are not economically sound. In several cases under observation, nephritis and gout have developed following the use of excessive amounts of cod-liver oil. There are definite limits to the beneficial use of oils containing vitamins.

Intramuscular injections of wheat-germ oil are contra-indicated from a commercial standpoint. When used in excessive amounts (intramuscularly) wheat-germ oil becomes walled off and remains unabsorbed.

The demand for light-colored egg yolks has been a potent influence in the reduction of the green feed intake of high-producing birds.

Our work leads us to the opinion that light-colored egg yolks as marketed at this time are indicative of an improper and possibly a dangerous nutritional balance.

The taking of blood-samples lessens egg production for a short period.

Selective breeding, increased production, continuous housing and impaired nutrition have markedly reduced disease resistance. Increased production has doubled or trebled certain nutritional requirements.

The high-producing bird whose eggs are hatched should receive added supplements to insure a more disease-resisting chick.

It is our belief, based upon clinical observation and our work, that cod-liver oil and cold-pressed wheat-germ oil, when

properly added in reasonable medical doses to basic foods, are important factors in reducing the incidence and mortality of fowl leukosis.

The beneficial dose of cod-liver oil and wheat-germ oil is not constant. The dose depends upon the character of the feed, severity of the disease, condition and age of the birds, and several other factors. Until a more scientific method is devised, the dosage in actual practice is best determined by clinical observation.

We believe that the nutritional treatment and care of the maternal bird offers, at least, one favorable line of attack on lessening the incidence of fowl leukosis.

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DISCUSSION

DR. E. E. JONES: I would like to ask Dr. Butler upon what basis he made a clinical diagnosis at the time of injection of the wheat-germ oil. What are the symptoms that call for blood-counts?

DR. HUGH HURST: What was the amount of cod-liver oil and the potency of the oil used in the commercial feeds on which these birds had been fed prior to the time that they were injected with the wheat-germ oil?

DR. BUTLER: We made a blood-count of some of these birds which showed evidence of myelogenous leukemia. Naturally we also made postmortems which substantiated our clinical diagnosis. We have found iritis, paralysis, and large liver, all in one flock.

I am not able to give you the potency of the cod-liver oil added by the manufacturer to commercial feeds. They do not give you the particular amounts. Many manufacturers do not make public their feed formulas. The feeds used in Montana in most of these flocks which we studied contained sardine oil, which was of low potency as far as vitamins A and D were concerned. Later some of these feeds were made to contain cod-liver oil in a percentage of about one-quarter of 1 per cent. One manufacturer, following our suggestion, added a cod-liver oil which is standardized, and contains 500 units of vitamin D and 3,000 units of vitamin A to the gram. This manufacturer endeavors to arrange his sales program so that the purchasers of his feed receive only freshly prepared feeds from rancid supplements.

Sires Affect Milk Production

Seventeen Jersey sires loaned by the Jeanerette (La.) Experiment Station of the U. S. Bureau of Dairy Industry to coöperating farmers show how good sires in farm herds transmit to their daughters the ability to produce more milk and butter-fat.

The 17 sires were bred to 297 cows, with an average annual production of 6,246 pounds of milk and 296 pounds of butter-fat, calculated to a mature basis. The daughters produced an average of 6,845 pounds of milk and 328 pounds of butter-fat. This is a net average of 599 pounds of milk and 32 pounds of butter-fat in favor of the daughters.

As in all proved sire work, some of the bulls failed to transmit production ability. Only four of the 17, however, failed to raise the average production.

One bull, placed with the State Normal College, Natchitoches, La., sired 14 daughters that produced 1,549 pounds more milk and 109 pounds more butter-fat than the dams.

Federal Meat Inspection Gets Popular Approval

Veterinary inspectors in the U. S. Bureau of Animal Industry report a general voluntary compliance with the federal meat inspection law. The packing industry knows that government meat inspection service is not only a valuable asset, but that failure on the part of any plant to comply with regulations may mean withdrawal of inspection. This in turn prevents a packing plant from doing business beyond state boundaries.

The Bureau reports that, because federal inspection is only for plants that have an interstate or foreign trade, there is a very noticeable trend for some states and cities to set up inspection systems of their own. Such inspection usually is patterned after the federal service and has the coöperation of the federal government.

Federal meat inspection—established in 1890 largely because of the foreign trade situation—now covers about 70,000,000 animals annually, or about two-thirds of the country's meat supply.

The Nature of Leukemia: A New Fundamental Principle Leading to the Development of Specific Disease*

By M. W. EMMEL, *Gainesville, Florida*
Florida Agricultural Experiment Station

The first cases of leukemia were reported in man almost simultaneously, but independently, by Craigie and Bennett,[1] in Scotland, and by Virchow,[2] in Germany, in 1845. Virchow called the disease "leukemia." The cause and nature of the disease have remained unknown.

Leukemia is characterized by hyperplasia of hematopoietic tissue, resulting in the prevalence of immature and often degenerative white blood-cells in the peripheral circulation and in the abnormal infiltration of these cells into the interstitial tissues of certain organs. It is a progressive and fatal disease.

Leisering (cited by Hutyra and Marek[3]), in 1858, apparently was the first to observe leukemia in animals. The disease has since been reported in almost every domesticated animal and, in a few instances, in wild animals.

The early history of leukemia contains many conflicting theories as to etiology. Traumata originating in the region of the spleen were considered a possible cause. A hormonal theory was advanced that disease of the endocrine glands exerted a stimulating influence on hematopoietic tissue. The idea was advanced that leukemia was in the nature of malignant tumors. As early as 1864, Virchow[4] conceived the idea that the disease was of infectious origin, but was unable to demonstrate the inciting agent. Various microörganisms, including those of the paratyphoid group, often were associated with the occurrence of the disease by early investigators. However, all of these organisms were regarded later as being devoid of etiologic significance.

The classical experiments of Ellerman,[5] in 1908, were the first to show that leukemia in the fowl could be transmitted to healthy birds by means of the injection of tissue emulsions and filtrates of affected organs. Subsequently, other investigators have shown that leukemia in the mouse, guinea pig and red squirrel can be transmitted by the injection of emulsified leukemic tissues or cells but not by non-cellular agents.

Due chiefly to their susceptibility, the chicken and mouse have become the principal animals for experimental leukemia investigations. MacDowell and Richter[6] have conducted extensive genetic studies in the mouse and the former developed a pedigreed "leukemia" strain, in which 90 per cent of the mice in the 18th and 23rd generations, surviving more than six months, developed leukemia spontaneously. Since leukemia in the chicken can be transmitted by a filtrable agent, many investigators have regarded this agent as a filtrable virus. Since the transmission of mouse leukemia requires a "cellular agent," mouse leukemia has been considered to be of a different nature than fowl leukemia.

Investigations of the author[7-13] have shown that leukemia in the chicken, dog, monkey, hog, goat and sheep can be induced by bacteria of the paratyphoid and typhoid groups. In the chicken, infection is dependent upon and varies with five factors, the interrelationship of which makes it possible for a single species of paratyphoid organism to induce ten distinct pathologic manifestations and typhoid species to induce two manifestations, which collectively are known as "leucosis" or the "leucosis complex." While often several manifestations are complicated with one another, each may be induced singly. In the other laboratory animals, paratyphoid species induce the leukemic leukemias, the

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

total white blood-count being above normal. Typhoid species typically induce the aleukemic leukemias, the white blood-count being normal or below normal. In chickens, leukemia induced by the paratyphoid species is "transmissible" after the experiments of Ellermann. The typhoid species do not induce typical fowl leukemia but induce aleukemic manifestations which are "non-transmissible." Aleukemia manifestations have been induced by *Eberthella typhi*, a variant of *E. typhi* and *Shigella dysenteriae*, while the leukemic manifestations have been induced by *Salmonella aertrycke*, *S. enteritidis*, *S. schottmülleri*, *S. typhimurium*, *S. psittacosis*, *S. pullorum*, *S. suipestifer* and *S. anatum*.

The microörganism must establish itself in the intestinal tract, from which it must enter the blood-stream over a period of time *but of insufficient intensity to induce septicemia*. During the period of infection, phagocytes destroy the invading organism. Endotoxin of the organism tends to inhibit agglutination and phagocytosis and is definitely destructive to blood-cells. Accumulations of endotoxin within the phagocyte, as a result of the liberation of endotoxin from the phagocytized bacterial cell, destroy the phagocyte. The continued destruction of phagocytes during the period of infection results in the intoxication of the blood-stream by the products of degeneration of the host's own tissues.

There is evidence to indicate that in many cases the invading microörganism tends to localize in the liver and spleen, with the result that these organs receive the brunt of the intoxication process. In case the impetus given by the microörganism is of sufficient intensity (a sufficient number of causal microörganisms over a sufficient period of time), a chronic but progressive, self-perpetuating autolysis of blood-cells is initiated.

During the period of infection the experiment animal need show no symptoms of illness. In chickens infection results almost immediately in hemocytoblastosis, which may be described as a leukemic state of the blood, but which need not become differentiated into one of the leukemias until two to eight months later, when definite symptoms of leukemia appear. In animals other than the chicken, infection results in leukocytosis, followed by a partial return of the blood-picture to normal, with the appearance of one of the leukemias two to eight months later.

After the development of one of the leukemias, the process of autolysis, which now has gained considerable impetus, proceeds as before, death eventually resulting from the complete intoxication of the body by the products of degeneration of the host's own tissues and an exhaustion of the hematopoietic tissues.

The causal microörganism stimulates autolysis to the extent that autolysis becomes a self-perpetuating process, then disappears from the host; thus, the microörganism cannot be isolated at death unless perhaps in rapidly fulminating cases. In case the microörganism fails to stimulate autolysis to a point at which it becomes a self-perpetuating process, recovery occurs, in many instances the host never having shown symptoms of illness. In the chicken, however, fowl paralysis, lymphomatosis and leukemic lymphocytoma may develop during recessive tissue autolysis.

A number of characteristic degenerative changes are observed in the blood-cells during autolysis; these changes are more pronounced in acute than chronic cases. Vacuoles of variable size often occur in the cytoplasm and nuclei; in the cytoplasm they usually are round, while in the nuclei they usually are elliptical. Pyknosis may be observed, particularly in the lymphocytes. "Budding," which is probably a result of altered surface tension, is an indication of degeneration and occurs in the lymphocyte and monocyte, and occasionally in the myelocyte.

Variations in staining reactions often are observed in blood-smears from animals in which the process of autolysis is particularly active. Liquefaction necrosis occurs in all types of cells; in the leukocytes the nuclei usually become involved first, necrosis then spreading to the cytoplasm as the cell enlarges, becomes distorted and finally ruptures. In the chicken the cytoplasm of the erythrocyte often becomes involved in liquefaction necrosis before the

nucleus and may totally disappear, leaving an enlarged, degenerative, eosin-staining mass, formerly the nucleus. In the chicken the rods of the rod-bearing eosinophile break into granules in typically leukemic birds. The eosinophile of other animals often contains fewer granules than the normal cell and the granules may be somewhat irregular in size. All of these types of degeneration are variable in individual animals and may occur in any combination.

Leukemia has been induced in the chicken, monkey and dog by the repeated injection of heat-killed causal microörganisms, which excludes the theory that the living microörganism is a vehicle for a filtrable virus. Repeated intravenous injections of benzene, phenol and xylol have resulted in the development of leukemia in the chicken. Leukemia has been induced also in the chicken, monkey, rabbit, dog, goat and mouse by the repeated injection of normal, freshly emulsified desiccated and autolyzed homologous tissues.

Suboxidation, as it occurs in many ill-ventilated, indoor-hen-battery establishments, leads to the development of leukemia in the chicken and is the primary cause of the major mortality problem under these conditions. Whether autolysis is initiated by living or killed bacteria, chemicals, homologous tissues, or suboxidation, the progressive nature of tissue autolysis resulting therefrom and leading to the development of leukemia is basically similar in all instances.

The "transmission agent" of fowl leukemia is regarded as the accumulated products of degeneration of the host's own tissues and not as a filtrable virus. The susceptibility of the chicken to leukemia permits the transmission of the disease by a filtrable agent. The interspecies transmission of leukemia is not possible, even in the chicken, since the autolysis of blood-cells is more easily initiated by the products of tissue degeneration of the species' own tissues than by those of another species.

Many noted investigators have considered leukemia to be of a neoplastic nature and have regarded the disease as a carcinoma of the blood. In the words of Richter and MacDowell[14]:

> There is so great a resemblance between the data obtained from studies of leukemia and neoplasms in animals that little doubt exists in the minds of contemporary workers in this field that the processes underlying the various neoplasias and leukemia differ only in so far as they involve different tissues.

These authors summarized the points of analogy to the following extent: (1) Heredity has a similar influence on the incidence of leukemia and carcinoma. (2) Leukemia in the fowl is transmissible by a filtrable agent resembling the transmissibility of the filtrable tumors in this species. (3) Leukemia and tumors in other animals are transmissible only by living cells. (4) In normal animals they are transmitted only to those of the same species. (5) Increased virulence is manifested after repeated transfer. (6) In both instances cell metabolism is altered. (7) In both instances early splitting of the chromosomes is observed during mitosis and the main differences between leukemia and neoplasms are related to the greater independence and motility of leukemia cells because of the tissue affected. To these, the present author would add that multiplication and degeneration of cells, so essential to the basic process of self-perpetuating tissue autolysis, is characteristic of blood-cells in leukemia, and of fixed tissue cells in the case of malignant neoplasias.

Therefore, it seems quite logical that the basic process of self-perpetuating tissue autolysis, which is the fundamental principle underlying leukemia, whether it be induced by any one of a number of inciting agents, furnishes a new and definite approach to the study of neoplasias of fixed tissue. In fact, the author has accumulated some evidence to show that leukemia, in the enlarged spleen of myelogenous leukemia in animals, is in the nature of a carcinoma.

SUMMARY

The new fundamental principle of self-perpetuating tissue autolysis has been described as the basic process involved in experimental leukemia in the chicken, dog, monkey, hog, goat and sheep. Tissue au-

tolysis leading to leukemia has been initiated by the intravenous injection of living and heat-killed species of paratyphoid and typhoid groups in all of these species; freshly emulsified, desiccated and autolyzed homologous tissues in the chicken, dog, monkey and goat; chemicals (benzene, phenol and xylol), and suboxidation in the chicken; the fundamental nature of the process was similar in all instances. The analogies of leukemia and carcinoma are enumerated and the hypothesis is advanced that the principle of tissue autolysis, which concerns blood-cells in leukemia, is involved in carcinoma, in which the process is confined to cells of fixed tissue.

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Public Hearing Announced on Food, Drug, Cosmetic Act

An informal public hearing to consider proposed regulations for the enforcement of the new federal Food, Drug and Cosmetic Act has been announced by Acting Secretary of Agriculture Harry L. Brown. The hearing will take place at 10:00 a. m., November 17, in the auditorium of the Department of Agriculture South Building, Independence Avenue between 12th and 14th Streets South West, Washington, D. C.

While the Act does not require the holding of hearings on all regulations, the Acting Secretary said, the Department desires to have the benefit of suggestions and constructive criticisms from consumers, interested industries, and others before these regulations are formulated for promulgation.

Those who are unable to attend the public hearing are invited to submit their expressions by letter not later than November 24, 1938. Copies of the proposed regulations may be obtained on application to the Food and Drug Administration, 4803 South Building, Department of Agriculture, Washington, D. C.

Congress Report

Dr. E. Gräub, general secretary of the Thirteenth International Veterinary Congress, is still receiving orders for the General Report of the Congress held in Zurich-Interlaken, Switzerland, in August. The General Report will contain all reports made to the Congress, with the discussions. The number of copies will be limited to the number of advance orders. The price of the General Report is 30 Swiss francs (about $7.00). Orders should be sent to Dr. E. Gräub, General Secretary, Seilerstrasse 23a, Berne, Switzerland.

A Study of the Blood in Horses Infected With Infectious Anemia*

By H. L. HAMMERSLAND, H. S. HERRIN *and* C. F. HAYNES
Montana Livestock Sanitary Board Laboratories, Helena, Mont.

Infectious anemia in horses has recently been reviewed by Gochenour[1] and others, the history of which has been thoroughly discussed, and reveals that this disease is still of great economic importance.

Records show that infectious anemia was diagnosed and reported in Montana before the year 1909. Since that time, numerous cases have been definitely diagnosed in various sections of the state under the supervision of the Livestock Sanitary Board laboratories and the veterinary research laboratories. The latter was able, through animal inoculations, to definitely diagnose infectious anemia in the majority of the cases cited. During our study, these cases were of a subacute nature.† Temperature charts have been kept, through the courtesy of the owners, since January 1, 1937. The temperatures ranged from normal to 108° F. at spasmodic intervals. These horses were wormed four times in 1937 and 1938 for bots, sclerostomes and cylicostomes. This was done in order to eliminate any factor which might change the blood-picture.

Blood Sedimentation Test

Blood sedimentation is more rapid in disease than in health, but such a test is not diagnostic of any one disease. The sedimentation test is, however, useful in differential diagnosis. For this reason, we have used it in connection with our blood problem in infectious anemia, not as a diagnostic agent, but as a step in our experiment. A normal or slowed sedimentation rate is usually not indicative of infection. Generally, the rate of velocity remains increased even after temperature, leukocyte count and general symptoms have subsided. This is of some value in infectious anemia, as most of our experiment animals showed no outward symptoms of the disease at the time the work was carried on.

In reviewing the current literature relative to sedimentation tests, we quote Jores and Strutz.[2] They point out that, since the formed elements of the blood, as well as some of the constituents of the plasma are subject to rhythmic fluctuations, it was to be expected that the sedimentation speed of the erythrocytes would likewise undergo rhythmic changes in the 24-hour period. They made investigations on persons with normal sedimentation values as well as on some with abnormal values. In those with normal sedimentation, it was found that, if the withdrawal of the specimens is begun in the morning, the curve shows at first a decreasing tendency. Beginning with 10:00 a. m., the curve shows an upward trend, which reaches its maximum at about 4:00 p. m. Then there is a slight downward trend until 8:00 p. m., which in turn is followed by a slight and brief upward trend, to be followed again by a downward trend which reaches its lowest point at about 6:00 a. m. In the cases showing abnormal values of the sedimentation speed, similar 24-hour curves were observed.

The authors discuss the various factors that might play a part in the development of the rhythmic changes. They do not think that the food intake or physical exercise exerts any influence. Moreover, they were unable to corroborate the observations of Imanow and Basilewitsch, according to whom the gastric motility influences the sedimentation speed. In further studies, they found that the examination of different patients on the same day resulted in similar curves, whereas examination of the same patient on different days resulted in different curves. They reached the conclusion that, in the practical employment of the sedimentation test, it is important to pay attention to the time of day at which

*Received for publication, May 27, 1938.
†Plans are being made to continue this study, especially on more acute cases.

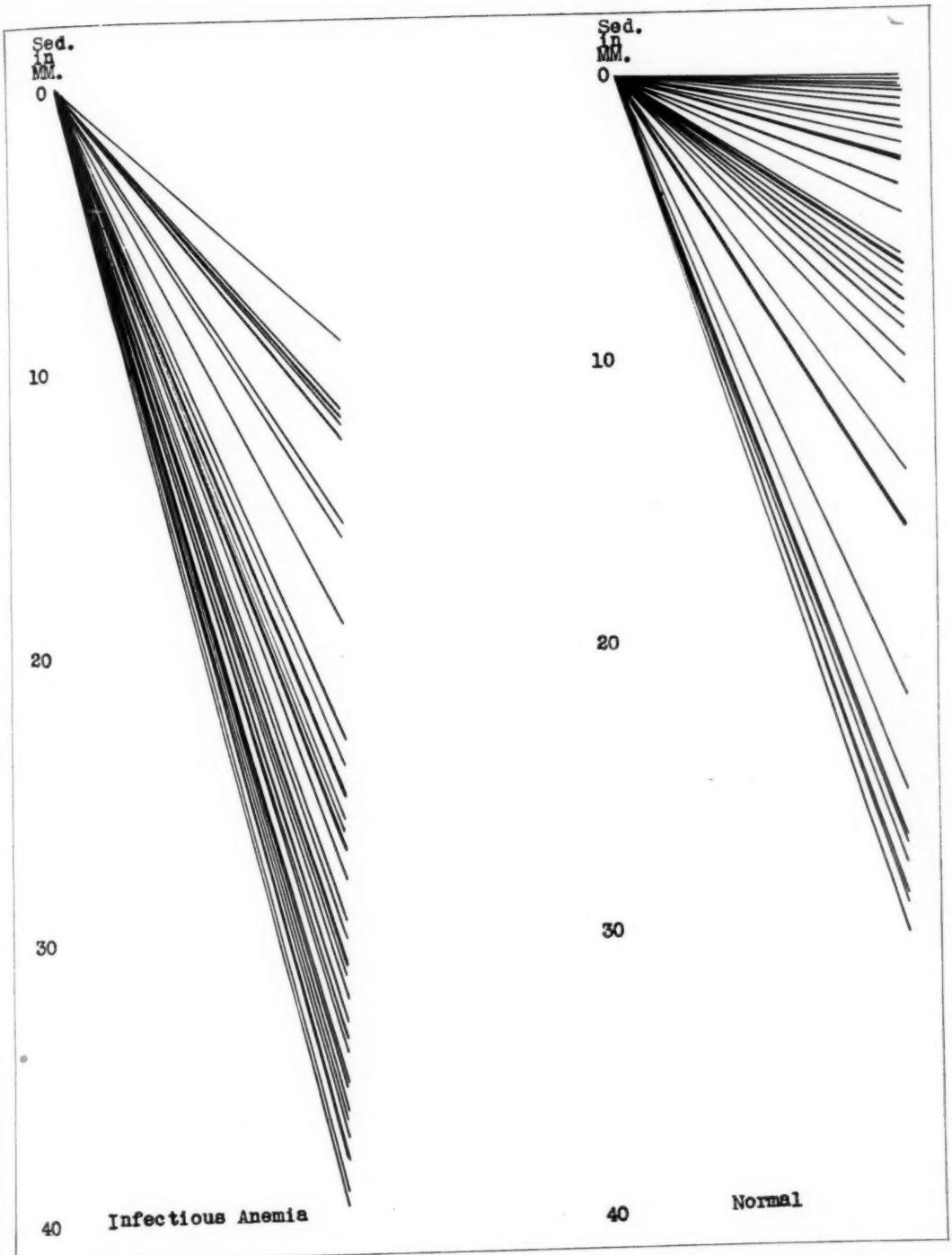

Fig. 1. An average sedimentation graph of infectious anemia. Maximum settling in 60 minutes. (60 normal horses and 40 affected with infectious anemia.)

the specimen is withdrawn, for a sedimentation value that is pathologic in the case of a morning specimen may still be normal if it concerns an evening specimen. Moreover, the authors observed two cases in which the sedimentation values were normal in the morning, but pathologic in the evening. In both instances this proved to be of diagnostic significance. One patient had a lymphogranulomatosis, the other a postanginal sepsis. In the latter case, the pathologic sedimentation values that appeared in the evening hours indicated a latent infectious process.

We may state that all of our horses infected with anemia were bled in the morning between 8:00 and 10:00 o'clock, after fasting and resting for twelve hours. It is evident, according to the above authors, that latent infectious processes might not show up at this period of the day. All of the horses were bled in the morning, but most of the controls were bled in the afternoon, which may account for the low sedimentation average shown in figure 2.

Several different technics were discussed. Cutler's[3] method was adopted. Especially designed sedimentation tubes of 1-cc capacity* were used with 12-cc Luer syringe and one stand, as described by Cutler. An area of 2 inches square was clipped in the region of the jugular vein and sterilized with a suitable antiseptic. One-tenth of 1 cc of a 3 per cent solution of sodium citrate was inserted into the Cutler tube by means of a 10-cc sterile syringe, using an 18-gauge needle three inches long. This was done to prevent the liquid from bubbling or adhering to the sides of the tube, so that an exact amount of fluid could be measured. One cc of blood was drawn from the jugular vein and the tube was filled to the zero mark. It was found advisable to use an 18-gauge needle, 1 inch long, in drawing the blood and an 18-gauge needle, 3 inches long, for placing the blood in the tube. This latter length needle aids in thoroughly mixing the blood with the citrate to prevent foaming. The tube was placed in the stand and kept

*American Journal of Medicine, May 1932, p. 643.

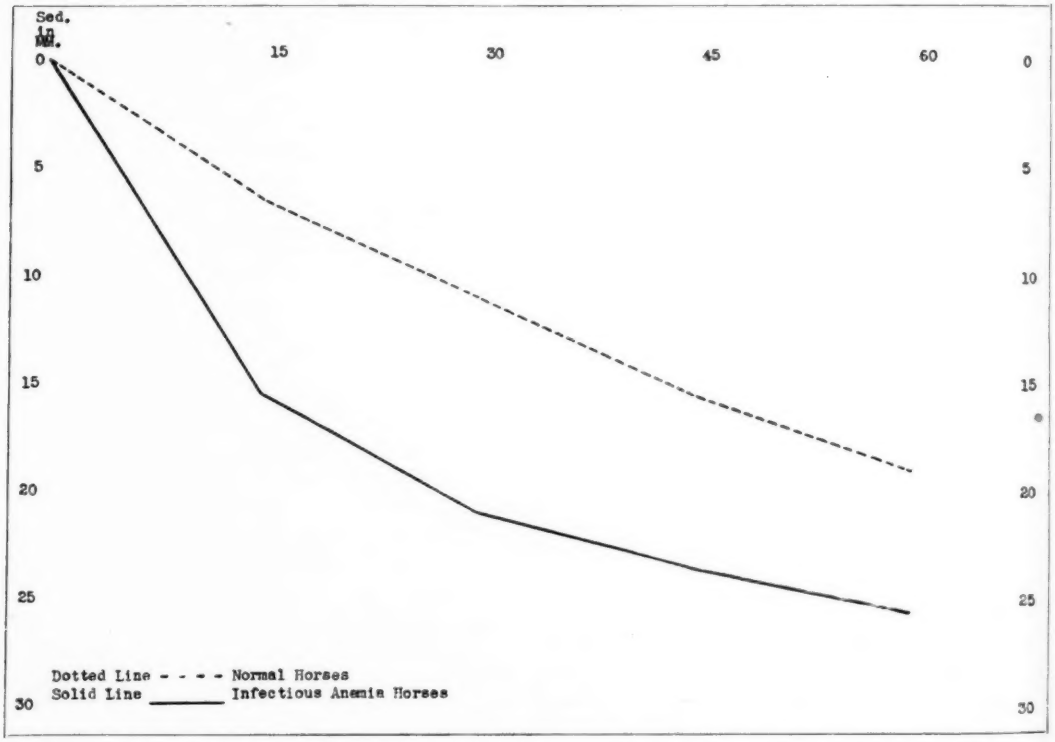

Fig. 2. An average sedimentation graph of infectious anemia. Time in minutes. (60 normal horses and 40 affected with infectious anemia.)

at a temperature ranging from 50° to 70° F. Temperature regularity is essential in the Cutler method, for extreme heat or cold alters the sedimentation rate shown on the graph. We found that 15-minute readings would give us approximately the same picture as the Cutler readings every five minutes.

BLOOD COUNTS

Blood was collected for complete blood counts at the same time that it was obtained for sedimentation and chemistry estimations. The blood examination consisted of a hemoglobin determination, a numerical count and a differential count. The Tallquist hemoglobinometer was used for all hemoglobin determinations. Toisson's solution was used as a diluting fluid for estimating the leukocyte and erythrocyte counts. Wright's method was used for staining all blood-smears. Table I shows an average of comparative results found in the blood of infectious anemia horses and that of normal horses.

TABLE I—*Blood counts.*

	HB.	LEUCOCYTES	ERYTHROCYTES	LYMPHOCYTES	POLYMORPHS	EOSINOPHILES	BASOPHILES	MONOPHILES
Infectious anemia horses	89%	10,120	4,780,000	46%	46%	6%	1%	1%
Normal horses	99%	8,240	5,854,000	52%	42%	4%	1%	1%

It is noticeable that there are no marked changes, but there is a slight leukocytosis and a slight anemia present. There were no changes in the blood cells, except in some of the more severe cases. In those cases, anochromasia and anisocytosis were present. Some megaloblasts were also found.

BLOOD CHEMISTRY

Blood for chemical examination was obtained from the jugular vein. A bleeding needle was used and the blood was put into sterile tubes. As some whole blood and some serum were used in the chemical tests, the blood was put into two sterile tubes, one containing 20 mg of potassium oxalate to prevent coagulation. About 10 cc of blood was placed in each tube.

The concentration of several constituents of the blood was materially altered for a time following feeding. Therefore, all of the horses tested in this experiment were taken off feed twelve hours previous to bleeding, in order to obtain uniform results. No preservative was used as chemical examinations were made in all cases within 24 hours after bleeding.

Todd and Sanford[4] was used as a reference in the chemical analysis. Table II shows complete results on the chemical tests made.

The following is a table of the methods used:

Test	*Method*
Urea nitrogen.	Folin and Wu.
Chlorides.	Wilson and Ball.
Dextrose.	Lewis and Benedict.
Calcium.	Clark and Collip.
Inorganic phosphorus.	Kuttner and Lichtenstein.
Iron.	Osterberg's modification.
Bile.	Gmelin.

The noticeable changes observed were an increase in calcium and a decrease in iron. A greater number of tests showed the presence of bile.

SUMMARY

1. The average sedimentation reading was 26 for the infectious anemia horses and 18 for the normal horses. We believe that the blood sedimentation test is useful in the diagnosis of this disease and has two values, one as a diagnostic lead and the other as a diagnostic gauge.

2. A slight leukocytosis was present together with a slight decrease in hemoglobin and the number of erythrocytes.

TABLE II—*Chemical tests.*

TEST	INFECTIOUS ANEMIA			CONTROLS		
	VARIANCE		AVERAGE	VARIANCE		AVERAGE
Mg. urea nitrogen in 100 cc of blood	5.3	32.6	13.4	8.9	12.9	10.9
Mg. chlorides in 100 cc of blood	284.4	376.8	335.4	309.3	357.6	333.8
Mg. dextrose in 100 cc of blood	31.4	91.3	56.7	53.5	80.0	65.8
Mg. calcium in 100 cc of serum	8.0	32.0	16.7	7.4	12.0	10.1
Mg. inorganic phosphorus in 100 cc of blood	2.6	6.5*	3.55	1.6	6.0	3.38
Mg. iron in 100 cc of blood	8.0	35.0†	21.3	23.3	30.0	26.7
Per cent bile present			39.3			10.0

*1 Impossible to read.
†5 Impossible to read.

3. A slight increase in calcium, and a decrease in iron were found. A large number showed the presence of bile.

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[5]Cutler, J. W.: The practical application of the blood sedimentation test in general medicine. Amer. Jour. Med. Sci., clxxxiii (1932), p. 643.

[4]Todd, and Sanford: Clinical Diagnosis by Laboratory Method. (8th ed., W. B. Saunders Co., Philadelphia, 1935.)

Foot-and-Mouth Disease in Foreign Countries

The unusually severe and extensive epizoötic of foot-and-mouth disease in European countries, which began about 17 months ago, still continues. After diminishing somewhat in the spring, incidence again has increased during recent months and at present the disease is widespread in many parts of the Continent except the Scandinavian Peninsula and some of the small countries, particularly on the east side of the Baltic Sea.

The writer availed himself of opportunities, during his travels in Europe this summer, to observe the disease in several of the countries, especially on dairy farms, in slaughter-houses and at experiment stations. Every zoölogical garden visited was posted with warnings regarding foot-and-mouth disease placed in front of all enclosures containing cloven-footed animals and, as a further precaution, additional fences were constructed ten feet from the pens to prevent contact with visitors. At creameries and cheese factories cocoanut-fiber foot-mats, soaked in disinfectants, were placed at all entrances to these establishments.

Great Britain and Sweden have been successful in controlling and reducing infection almost to the point of complete eradication but occasional outbreaks have intervened. The same is true of Denmark and Switzerland and the latest reports from the latter country show the disease has been confined to the canton of Geneva, which is contiguous to France on three sides. Beginning in August, there has been a marked increase in the number of infected herds in Belgium and the Netherlands. In central and southern Europe the extensive spread has continued. More than 45,000 herds became infected in France in August, while Germany reported more than 130,000 infected premises and Poland more than 100,000. In Czechoslovakia, Yugoslavia, Hungary and Italy the prevalence apparently remains unchecked.

After apparently eradicating one outbreak of foot-and-mouth disease last winter, the Union of South Africa had a very serious outbreak in June, necessitating the destruction of many thousands of cattle. A further outbreak in another region occurred in August, involving about 20,000 head.

Conditions in other countries and continents remain the same as for many years past. North and Central America, the West Indies, Australia and New Zealand, and Venezuela and Colombia continue to be free from foot-and-mouth disease. Other South American countries still harbor some infection and the disease is known to exist in practically all countries of Africa and Asia except Japan. J. R. M.

PRENATAL FRACTURE OR FETAL ANOMALY—WHICH?*

By ROY E. NICHOLS, *Columbus, Ohio*
College of Veterinary Medicine,
Ohio State University

On January 13, 1938, a day-old, 25-lb. purebred Jersey heifer calf was presented at the Veterinary Clinic of the Ohio State University with the following history: according to the owner's records, the dam should have calved one month later. He did not see the cow give birth to the calf, but found it in the gutter in the morning. He noticed that the left rear leg was dangling and thought that the dam had stepped on it.

Upon examination, a definite separation of the left tibia near its center was palpated and, immediately lateral to the distal end of the proximal portion of the bone, a small wound was noticed. The ends of the bone could be made to crepitate. The wound did not have the appearance of a fresh wound, but showed a fairly wide zone of new, healthy-looking skin around a small red center. By pressure at the sides of the wound, a small amount of white, cheesy material, similar to that found in a sterile abscess, was expressed.

Further palpation revealed a lack of uniformity in shape of the two free central ends of the tibia. The distal end of the dorsal portion felt smaller than the dorsal end of the ventral portion. It was also noticed that the affected leg was a little shorter than the right one and the tendons somewhat contracted. There appeared to be a lack of development of the musculature of the affected limb as compared with the normal one.

*Received for publication, March 29, 1938.

Because of the character of the wound and the asymmetry of the free ends of the tibia, it was decided to withhold diagnosis until an x-ray could be taken. Figure 1 shows the calf as presented. Figure 2 shows a medio-lateral x-ray view of the affected part and figure 3 an antero-posterior x-ray view. Note the lack of symmetry of the central ends of the affected bone.

After examination of the x-ray pictures, can one call the condition a day-old fracture? If a day-old fracture, how can one account for the zone of healing about the wound, the cheesy material coming from the wound and the lack of symmetry of the affected ends of the bone? If not a fresh fracture, could the condition have been a prenatal fracture, the wound resulting from pressure on the skin over the pointed distal end of the dorsal portion of the tibia and the asymmetry of the affected ends of the bone resulting from variations in pressure upon them? Or could the condition have resulted from faulty development of the bone in that region, the wound resulting from the pressure?

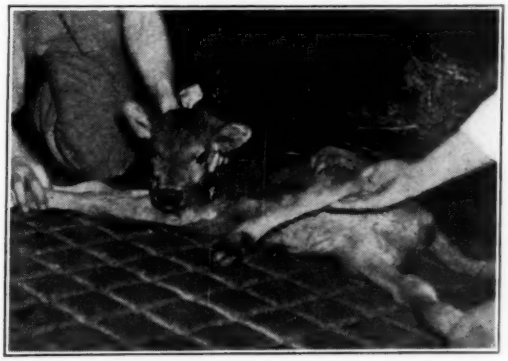

Fig. 1. The calf as presented for treatment.

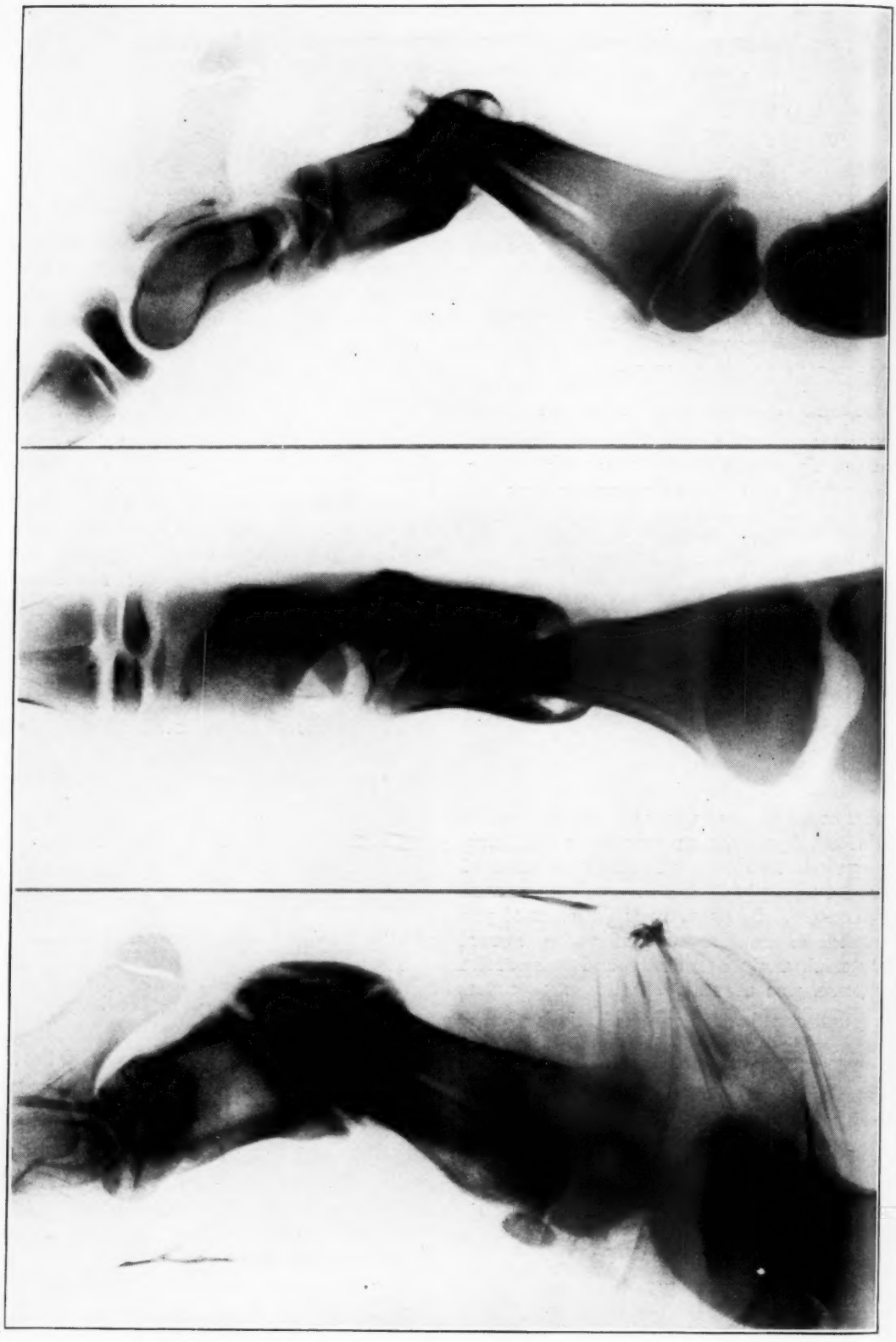

Fig. 2 (above). Medio-lateral x-ray view of the affected part.
Fig. 3 (middle). Antero-posterior x-ray view.
Fig. 4 (below). X-ray picture showing new bone about the affected region.

Despite the fact that a diagnosis of fracture could not be made conscientiously, it was decided to treat the condition as such. A modified Thomas splint was applied for traction, as in small animals (fig. 5). However, not so much traction could be applied

Fig. 5. Calf with modified Thomas splint applied.

as in small animals because the fold of the flank in calves extends more ventrally than in dogs or cats. The animal was up and moving about with ease after the second or third day. The wound was completely healed in four or five days. Because the animal had not put weight on the claws of its affected limb, it was decided to place the leg, as soon as it was thought to be

Fig. 6. Calf just before its discharge from the hospital.

safe, in a yucca board splint, let the calf limber up its muscles and tendons, and bring its claws into wear. This was done on the 14th day. Within a few days, the animal was bearing considerable weight upon the limb. Frequent palpations and an x-ray now and then revealed considerable callous tissue about the area. Figure 4 (x-ray) shows new bone about the region just before the splint was removed. On the 23rd day, the splint was removed and from then on the animal moved about with comparative ease. The leg was somewhat stiff and a bit shorter than the other, but no doubt the animal will turn out to be quite a useful cow. Figure 6 shows the calf a day or so before its trip home. As may be seen, most of the weight of the rear limb is placed on the affected leg.

If the condition was a prenatal fracture, why was there little or no evidence of healing of the ends of the bone at birth? If a fetal anomaly, why did the bone ends knit so well after birth? Perhaps motion of the parts brought about by the playful antics of a well-fed, healthy calf was all that was needed to stimulate new bone growth.

Prenatal fracture or fetal anomaly—which?

A HERD CONDITION OF SWINE CHARACTERIZED BY ICTERUS AND ANEMIA*

By A. H. QUIN, JR., *Des Moines, Iowa*

On many occasions during the past 20 years, the writer has noted a herd disease of growing shoats characterized by splenic enlargement, generalized jaundice and coincidental anemia. It occurs only during the summer months and, to the best of our knowledge, is seen only in shoats ranging from 40 to 100 pounds in weight. It has been noted in both immune and non-immune herds, but is more common in the former.

Available literature contains practically no references to this disease other than a report by Doyle,[1] of Purdue University, made in 1932.

Cases of this ictero-anemia may appear rather suddenly and involve a variable percentage of the total herd. Death losses from the herd standpoint rarely exceed 20 per cent, but the mortality rate, as it affects individual animals, is very high.

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SYMPTOMS

The onset of this condition is quite rapid and death may occur in from 24 to 72 hours after the initial symptoms are noted. Inappetence, increasing prostration, stiffness, "thumpy" respiratory movements, constipation, and a temperature range of 104° to 106° F. are constant. Inspection reveals marked icterus of the eye membranes and mouth as well as the skin. Very commonly a dry, hacking cough is heard.

LESIONS

The autopsy findings of pigs affected with this piroplasma-like icterus are so constant that they may well be considered pathognomonic.

As the shoat is bled, one may readily see the anemic character of the blood as well as the iridescent reflection of greenish tints. One notes that the volume of the bleeding is markedly decreased. All of the subcutis, fascia and serosa exhibit marked jaundice.

The lymph-glands are edematous and icteric, while the pericardial sac and the pleural and peritoneal cavities contain an excess of yellow-tinted fluid.

The liver commonly shows a yellowish color with white, blotchy areas on the parietal surface. The bile-ducts are patent and unoccluded by parasites.

However, the most constant and striking finding is the greatly enlarged, soft, pultaceous spleen. Very often the spleen of a 60-pound shoat will exceed in size the normal melt of a full-grown sow.

The intestines show little if any pathological change other than icterus, while the lungs, in the main, are free from lobar pneumonic changes. However, the lungs may show some intralobular edema. No septicemic changes are seen unless the condition is noted in pigs during the reaction stage of serum-virus usage.

The kidneys show some evidence of involvement, such as adherence of the capsule and a darker than normal color.

DISCUSSION

Some practitioners have expressed the opinion that this herd jaundice of pigs is of dietary origin. On the other hand, it has been encountered in herds on a wide variety of rations, either when pastured or yarded. The fact that it often appears in shoats, from two to six weeks after immunization against cholera, has led others to opine that it may be related to the cerebro-hepatic condition cited by Marsh and others following the use of encephalomyelitis serum on horses or diphtheria antitoxin in children. This premise is unlikely, for the disease is strictly seasonal and has been noted in herds never exposed to serum and virus.

The fact that Doyle demonstrated Rickettsia or anaplasma-like bodies in the red corpuscles of such pigs, points to the probably true nature of the trouble. This has been further substantiated by Dr. H. C. Smith, of Fort Dodge, Iowa, who also noted intracorpuscular bodies in the red cells of a native-raised Iowa pig showing splenomegaly and jaundice.

It is the writer's opinion that this icteroanemia is a vector-borne disease (mosquitoes, probably) and that the causal agent is a piroplasma or anaplasma.

Veterinarians should coöperate with commercial and college laboratories by submitting blood-smears from pigs so affected. Or, if they have laboratory equipment, they might try staining such smears with Giemsa or modified Wright stain.

TREATMENT

So far, no really effective method has been found for blocking losses of this type. On the presumption that the disease may be protozoan in character, a rather heavy dose of intramuscular sodium cacodylate is indicated and, even though non-specific, it serves as a powerful alterative and tonic. The dose should range from 20 to 45 grains on pigs weighing from 40 to 100 pounds. Fowler's solution, three ounces per ton of live weight daily, also is in order. This may be followed by a copper and iron tonic or by copper sulfate in the drinking water to correct anemia.

Quite often, herds with ictero-anemia carry heavy ascarid infestations and elimination of these parasites, of course, is always in order.

REFERENCE

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DEAFNESS DUE TO TOXICITY OF OIL OF CHENOPODIUM*

By JAMES FARQUHARSON,
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Division of Veterinary Medicine,
Colorado State College

On August 22, 1938, a four-year-old Brussels Griffon weighing 16 pounds was admitted to the veterinary clinic. In the course of routine examination the fecal material revealed the presence of ascarid eggs.

The dog was starved for 24 hours and then 1.5 cc of oil of chenopodium in capsules was administered, followed with a dose of castor oil. Vomiting occurred shortly after the administration of the oil of chenopodium. The next day it was noticed that the dog was deaf as he did not respond to call or noise. Otherwise he appeared normal. A careful history of the case was taken from the time of discharge from the hospital in an effort to determine the cause of such a condition. Due to close confinement in the house, nothing of any importance was ascertained. The next consideration was an attempt to determine whether the toxic effects of the oil of chenopodium might be responsible.

Oil of chenopodium is known for its toxicity. It causes general depression, nausea, vomiting, paralysis, stupor and coma. A review of available veterinary texts and literature pertaining to this subject fails to mention deafness as one of the unfortunate sequelae. However, human texts mention the fact that deafness is relatively common in man following the administration of oil of chenopodium in therapeutic doses. Quoting Sollmann in regard to the side effects of therapeutic doses in man, "Deafness occurs in 20 per cent. It varies in intensity from a disagreeable after effect to complete bilateral loss of hearing, lasting usually for a week to several months, but in rare cases still present after two years. (Roth, Oppikofer, 1919.)"

From this information it is difficult to believe that deafness is as common in animals as it is in man following the use of therapeutic doses of oil of chenopodium. With the universal use of this drug as a vermifuge in horses, pigs and dogs, certainly more cases of deafness would be reported. This would be particularly true in the case of the horse and dog that respond to call. However, it may not be generally noticed in pigs.

After two weeks, no improvement was shown in the hearing in this particular case. Except for the deafness, the dog appears normal and has gained in weight.

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A NOTE ON A CASE OF ERRATICISM IN STRONGYLUS EQUINUS*

By JOHN W. BRITTON, *Berkeley, Calif.*
Division of Veterinary Science, University of California

During the course of an investigation of parasitic gastritis in horses, a case of erraticism in *Strongylus equinus* was noted. The subject was a wild, nine-year-old range stallion from Winnemucca, Nevada. On opening the stomach preparatory to making scrapings to determine the presence of stomach worms, we discovered three large, soft nodules in the gastric mucosa averaging 21 mm in height by 15 mm in width. With the exception of a small, pin-point hemorrhage on the top of each nodule, there was no indication of any inflammatory reaction. When the nodules were cut into, each was found to contain a single active larval *S. equinus*, surrounded by a thick, cheesy exudate. The larvae were nearly mature, being provided with a buccal capsule and the three characteristic teeth of the species. They measured 19 to 21 mm in length.

Although the life cycle of *S. equinus* has never been satisfactorily worked out, Thwaite[1] and Taylor,[2] working independently, have indicated the usual sites of

*Received for publication, September 19, 1938.

larval invasion to be, first, in verminous cysts in the mucosa of the colon and, later, as agamic forms in the liver. Thwaite and Taylor both emphasize the finding of the larvae in the pancreas, lungs and perirenal tissues as common examples of erraticism in this species. The finding of viable agamic forms in gastric nodules seems to be another instance of erraticism in *S. equinus*.

The indications are that erraticism in *S. equinus*, as with the other members of the genus *Strongylus*, is more prevalent than is commonly believed. Erraticism in host-parasite relationships is very often more dangerous to the host than is the case when the parasite follows its normal course of development. Further, the diagnostic difficulties encountered in erraticism among parasites add to the importance of the condition.

REFERENCES

[1]Thwaite, J. W.: A contribution to the study of the parasitic life history of *Strongylus equinus*, Mueller 1780, together with a description of some lesions found in a donkey as a result of dosing with the infective larvae of this parasite. Ann. Trop. Med. & Parasitol., xxii (1928), p. 291.

[2]Taylor, E. L.: The third stage larva of *Strongylus equinus*. Ann. Trop. Med. & Parasitol., xxiv (1930), p. 545.

OPERATION FOR CONGENITAL CATARACT IN THE DOG*

By A. G. MADDEN, JR., *Madeira, Ohio*

Insofar as can be ascertained by the study of any literature, no cases of operative treatment for cataract in the dog have been reported. It is the purpose of this paper to describe the operative procedure carried out for such a condition.

In July of this year, a wirehaired fox-terrier, three years of age, was presented at our hospital with the following history: During the past two years, the owner had noticed a film over the dog's eyes and a diminished acuteness of vision. This condition had grown worse until the dog was totally blind. There was no history of trauma or disease of the eyes. Other history was essentially negative.

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Examination of the eyes revealed no inflammatory symptoms. Ophthalmoscopic (+15) examination showed a black opacity of the lens of the eyes against a red field. Practically the entire lens of each eye was involved. A diagnosis was made of congenital complete cataracts. The owner was informed of the diagnosis and told that any treatment other than operative would prove useless. Permission was given and the operation performed.

Operative procedure was carried out with the following instruments:

1. Eye speculum.
2. Lid elevator.
3. Fixation forceps.
4. Garafe knife.
5. Capsule forceps.
6. Cystotome.
7. Lens spoon.
8. Wire loop.
9. Curved iris forceps.
10. Curved iris scissors.

The patient was weighed in and 1 cc of nembutal given intravenously for each five pounds of body weight. The area around the eye was cleaned with tincture of green soap and painted with tincture of merthiolate. The patient was draped with sterile towels and a few drops of cocaine solution (10%) were instilled in the eye.

The conjunctival sac was flushed with a large quantity of warm saline. The eye speculum was inserted and the fixation forceps applied just below the cornea. Corneal incision was then made, which comprised about two-fifths of the circumference of the cornea in the plane of its transparent margin. The garafe was thrust into the cornea above the horizontal meridian, through the anterior chamber, and emerged at a point opposite the puncture. By pushing the knife upwards by a to and fro movement the section was completed in the same plane, terminating at the upper margin of the cornea. Following this an iridectomy was performed, making as narrow a colomba as possible. The capsule forceps were then introduced, closed, then opened, and as large a piece of the capsule removed as possible. The fixation forceps were then removed and the lens expelled

by pressing gently upon the lower part of the cornea, upwards and slightly backwards with the back of a Vaviel spoon.

The lens emerged through the corneal wound where it was received by the wire loop, its exit being facilitated by gentle depression of the posterior lip of the wound with the loop. The remaining portion of the capsule was then cleaned, thus avoiding the possibility of a blood-clot. A few drops of antiseptic solution were instilled into the conjunctival sac and the lids closed for a few minutes, after which the eye was inspected. Lens débris in the anterior chamber and in the wound was liberated by gently stroking the cornea upwards with the back of the Vaviel spoon. The iris was smoothed out. The cornea was replaced and sutured with 6-0 suture. A boric acid pack was then applied and, over this, dry absorbent cotton with a figure-of-8 bandage.

The dressings remained in place for seven days, after which time the bandages were removed and the patient was kept in a dark kennel for a few days, gradually permitting more light. The sutures were removed the tenth day and the eyes were cleaned daily with boric acid solution. At the end of four weeks, the patient was able to see well enough to play ball and notice objects at a distance with no difficulty whatsoever. At this time, the patient was discharged from the hospital, with instructions to the owner to return him each week for examination. At each visit his vision has shown improvement, with complete uneventful recovery.

HEMOLYTIC STREPTOCOCCUS INFECTION OF THE THROAT*

By S. W. STILES, *Falmouth Foreside, Me.*

The subject of this report is a five-year-old male Boston terrier, no different than the ordinary house pet, with the exception that this dog was fed a good, balanced diet.

This case to me was unusual, because of the temperature dropping to 95° F. and remaining there for approximately 48 hours, the comatose condition of the dog for 72 hours, and the subsequent rapid recovery in an apparently fatal case of infection.

The daily treatment and temperature readings were as follows:

8-17-38: Dog presented during office hours. T. 103.3° F., tonsils red and swollen, inappetence. Prescribed tincture of metaphen in glycerin to paint the throat every two hours and five grains of aspirin every four hours. Dog taken home and treatment followed.

8-18-38. Owner called. Dog about the same.

8-19-38: Dog brought to the office early this morning. T. 97.8° F., had two convulsions, throat badly inflamed, mucous membranes congested. Camphorated oil given intravenously, 5 grains of sulfanilamide every three hours, whiskey and water per rectum every two hours, and a heating-pad used around the dog. Throughout the day the dog was very quiet. Morning, noon and evening temperatures were 97, 95 and 96° F., respectively.

8-20-38: Same treatment was used, except 1/20 grain of strychnine was given every two hours. Patient very quiet, mucous membranes congested and cynotic, and throat bad. Prognosis, very bad. Morning, noon and evening temperatures were 98.6, 99.1 and 99.0° F., respectively.

8-21-38: Cream, whiskey and karo syrup were given by way of the mouth, along with the other treatment. The whiskey and cream per rectum were given every four hours. Patient was some better, but still quiet. Throat and mucous membranes have a better color. Temperatures during the day were 98.6, 99.1 and 99.0° F.

8-22-38: Strychnine was given every four hours and 2 grains of sulfanilamide morning and night. Patient seemed better, becoming restless. Ate some food and drank some water. All other treatment discontinued. Temperatures during the day were 99.6, 99.0 and 99.8° F.

From August 23 on, recovery was rapid and I was advised by the owner that the dog was well and healthy by September 1.

This case received excellent nursing from the owner, and it is due to this nursing that the dog recovered.

*Received for publication, September 22, 1938.

SAND IMPACATION IN A COW TERMINATED BY SUFFOCATION*

By EDWARD E. THOMPSON, *Roanoke, Va.*

On September 29, 1938, I was called to see a four-year-old purebred Guernsey cow that had been off feed, stiff and dull for two days. General examination revealed a temperature of 96° F., cold extremities, and weak pulse. The habitus was stiff, there was no inclination to move, and the lumbar region was arched. Rectal examination showed the abdomasum to be distended with fluid to a pear shape within 12 inches of the pelvic inlet and against the kidneys dorsally. On passage of the stomach-tube and the injection of a small amount of fluid, immediate symptoms of suffocation developed and the cow succumbed.

Postmortem revealed collapsed intestines with dry ingesta and a markedly distended abdomasum. Incision of that organ was obstructed for 4 inches with sand, and a quantity of sand was within the abdomasum. The omasum also contained considerable sand, and the mucosa of both organs was necrotic. Examination of the esophagus and respiratory organs showed that a bolus of food had been regurgitated, along with fluid, and had closed the laryngeal orifice.

"CANICOLA FEVER," A PROFESSIONAL HAZARD†

By K. F. MEYER, B. STEWART-ANDERSON *and* B. EDDIE

George Williams Hooper Foundation, University of California, San Francisco, Calif.

It is deemed imperative to call attention to the probability that the veterinarian, in the course of his professional duties, may become the victim of so-called "Canicola fever." In 1937, a veterinarian, who had a severe attack of jaundice followed by hemorrhagic nephritis about eight months previously, was anamnestically diagnosed as a special case of Weil's disease, since his blood-serum agglutinated the *Leptospira canicola* in a dilution of 1:300. Recently, an assistant veterinarian with a stormy, undulant fever-like disease presented a specific Canicola agglutination reaction in a dilution of 1:10,000. He was hospitalized and, on the 32nd day of his illness, *L. canicola* was isolated by guinea-pig passage from his urine. No doubt exists that he and, in all probability, the other patient have suffered from Canicola fever. Both men handled a great many jaundiced dogs, and some were affected with Stuttgart's disease. Since it is now proven that, in certain areas in California as many as 34 per cent of the dog population is subclinically infected with *L. canicola*, urinary shedders are doubtless quite common. Aberrant contact infections of man may, therefore, be anticipated. According to serum tests on dogs from the vicinity of New York, about 9 per cent are subclinically infected with *L. canicola* and about 2.8 per cent with *Leptospira icterohaemorrhagiae*.

The course of these infections in man is frequently atypical, complicated with meningitis and of long duration. Frequently, they are mistaken for influenza, undulant fever or tuberculosis. A serological examination of the blood and a bacteriological analysis of the urine may clarify the diagnosis. Dogs suffering from nephritis and dysuria should be examined for leptospirosis. They should not be returned to their owners, since the risk of causing human infections is by no means small. Epidemics of Canicola fever among men and dogs have been observed in Holland (Roos *et al*) and are probably also masquerading under different names in various parts of the United States. The George Williams Hooper Foundation of the University of California, The Medical Center, San Francisco, California, will gladly examine serum specimens. A detailed report of the dog disease will be published in the near future.

*Received for publication, October 5, 1938.
†Received for publication, October 17, 1938.

THE ANATOMY OF THE DOMESTIC ANIMALS. Septimus Sisson, S.B., V.S., D.V.Sc., late Professor of Comparative Anatomy, Ohio State University, Columbus, Ohio. Revised by James Daniels Grossman, G.Ph., D.V.M., Professor of Veterinary Anatomy, Ohio State University. 3rd edition, revised. 972 pages, with 770 illustrations, many in colors. W. B. Saunders Company, Philadelphia, 1938. Cloth, $12.00.

The revision of this well-known text represents a difficult achievement. "The general plan and scope of the book remain unchanged," as stated in the preface. Although there are a greater number of pages in the new edition, the paper is lighter, making a less bulky volume.

In reviewing the book, one observes the many worthwhile changes and some things that may be criticized in the interest of veterinary anatomical science.

A brief paragraph describing the pes was added on page 31. This is a logical and helpful change, but it repeats a statement made a few paragraphs previously.

There apparently was some effort made to eliminate foot-notes and portions printed in small type, either by inclusion in the main text or by eliminating them entirely. This was not done consistently, however. To cite an example: On page 55 of both editions, a paragraph on the "lateral mass" of the ethmoid was, in the revised edition, included in the main paragraphs, while on page 63 two paragraphs in fine print were retained as such, the second of which would appear to be of as much value to the preceding paragraph as the one on page 55 referred to above.

The use of contrasting colors on certain figures, as 48 and 50, has greatly enhanced their usefulness. One could have hoped that the same changes could have been made on other similar figures. Figure 575 appears in both the second and third editions. Contrasting distinctness of colors used in this figure is greatly decreased in the later edition. Many of the newly added figures are excellent and enhance the value of the textbook. Some of these figures, especially those that apparently have been made from photographs of dissections, however, do not compare in excellence with those of the second edition. In observing, for example, the peculiar curvature of the ribs in figures 359, 365 and 366, one is much disturbed by the departure from the natural.

Some things could be hoped for in labeling. In figure 368a the significance of the three numbers, "6,6,6," is quite vague. The legend gives the word "caecum." A departure from a system followed in former editions is observed also in figures like "Fig. 395*a*.—Cross Section of Shorthorn Cow." The titles of the figures in the earlier editions covered the regions concerned and would make for quicker identification. The diagram, figure 695, *a*, could have been labeled more completely.

The new discussions of the paranasal sinuses of the sheep and the additions of several figures from Baum's "Lymphgefassystem des Rindes" to the descriptive matter on the 'Lymphatic System of the Ox and Sheep" have increased materially the usefulness of the portions concerned.

Some brief introductory statements have been added to the section devoted to "The Sympathetic Nervous System." Since this textbook is mainly for student use, the value of the reference to Langley's work is questioned, as students would not have had occasion previously to learn about his classification. It would seem highly desirable that a more extended discussion of the so-called "Sympathetic Nervous System,"

based upon the work of present-day neurologists, be given. Such a discussion should attempt in greater detail to bring to the student the relations of the terminology of the neurologist to the disposition of this system in the domestic animals.

In these days, when there is such a great development of the small-animal field of veterinary practice, it would appear that the anatomy of the dog would merit a greater share of veterinary anatomy texts than formerly. With the exception of the addition of two figures in the digestive system, there are practically no changes, in this edition, on the anatomy of the dog.

A section of approximately 20 pages on the anatomy of the chicken has been added at the end of the book. For a brief discussion, the more important structures have been quite well described. The portion devoted to "Myology" is very brief. Probably a discussion of the abdominal muscles would have enhanced the usefulness of this section.

In so complete a text, it would appear that a few well-chosen references for the different sections would have added greatly to the serviceableness of the text.

This text book has no peer in its field in the English language. With the changes, it should make a valuable addition to the library of any veterinarian. In general the changes will enhance its usefulness to the student of Veterinary and Comparative Anatomy. For English-reading students in the field of Veterinary Medicine it is a veritable *sine qua non.*

H. L. F.

SECRETION OF MILK. Dwight L. Espe. 1st edition, xiv+265 pages, with 49 illustrations and 34 tables. Collegiate Press, Inc., Ames, Iowa, 1938. Cloth, $3.00.

The author is Assistant Professor in Dairy Husbandry at Iowa State College. The material presented, according to the author, is used in a course in milk secretion. The objective is to focus the training received by the student in anatomy, physiology and nutrition, on problems dealing with the normal functioning of the mammary gland.

The book is not especially designed for the practical dairyman or the research student. There are listed 695 references to the literature, included in which are none by the author. This assures the reader that the book is not designed to put across some pet notions of a college professor. The illustrations are excellent and the tables exceedingly instructive.

The book is divided into three parts: Part I deals with the phylogenetic development and the anatomy of the udder, both gross and histologic, as well as its development and involution. Part II deals with the theory of milk secretion, factors affecting its amount and composition, nervous and hormonal control, as well as miscellaneous factors related to milk secretion, such as the use of drugs. Part III has to do with the effect of feed on the amount and composition of milk, with reference to carbohydrates, fats, proteins, minerals, vitamins, taste, flavor, odor, drugs and hormones.

The publisher has done a good job of printing and binding. The author is to be congratulated on presenting in an excellent manner this well-written work on a subject of interest to both the student of milk production and the veterinarian whose practice is largely with dairy cattle.

W. G.

New Fort Dodge Branch

Fort Dodge Laboratories have announced the opening of a new branch at 1332 Marshall Street, N. E., Minneapolis, with Dr. John S. Dick, Jr., in charge. Increased business in Minnesota necessitated the opening of the Minneapolis office, in addition to the branch already located at Mankato, in charge of Dr. W. C. Cullen, Fort Dodge officials state.

There is music wherever there is harmony, order, or proportion.—Sir Thomas Browne.

Friendship is the shadow of the evening, which strengthens with the setting sun of life.—La Fontaine.

THE BACTERICIDAL ACTION OF PROSTATIC FLUID IN DOGS. Guy P. Youmans, Joseph Liebling and R. Y. Lyman. Jour. Inf. Dis., lxiii (1938), p. 117.

Prostatic fluid was collected aseptically from normal dogs and tested for germicidal action upon *B. coli, S. aureus,* streptococci and gonococci. A marked germicidal power was demonstrated for *B. coli, S. aureus* and streptococci and a very slight germicidal action for gonococci. Different samples of prostatic fluid varied considerably in their germicidal power. The fluid was tested for complement and lysozyme and neither of these factors could be demonstrated. The germicidal agent could be removed by absorption with either *B. coli* or *S. aureus* and was apparently nonspecific. The germicidal power was slightly reduced by heating at 50° C. for 30 minutes, reduced over 50 per cent at 55° C. and completely destroyed by heating at 60° C. for 30 minutes. The prostatic fluid could be kept in the ice-box for over three months without loss of potency. As tested, the germicidal power of dog prostatic fluid was far greater than that of the blood serum taken from the same animal. The nature of the germicidal substance has not been determined, but it appears to be similar to that responsible for germicidal effects in other body fluids and excretions.

VITAMIN C AND RESISTANCE OF GUINEA PIGS TO INFECTION WITH BACTERIUM NECROPHORUM. Norman B. McCullough. Jour. Inf. Dis., lxiii (1938), p. 34.

The author studied the relation of vitamin C deficiency in the guinea pig to infection with various strains of *Bacterium necrophorum.* Avirulent human strains that did not produce infection in guinea pigs on a vitamin C-adequate diet, produced minor abscesses in the extremely scorbutic guinea pigs. Animals with a subacute scurvy did not develop infection. Vitamin C therapy resulted in prompt recovery of infected scorbutic animals. The slightly more virulent animal strains which produced transient abscesses in the control animals gave a more severe degree of infection in the vitamin C-deficient guinea pigs. The lesions persisted and the guinea pigs died much sooner than did uninfected vitamin C-deficient animals. A severe scorbutus was necessary before a drop in resistance to *Bacterium necrophorum* became evident.

THE INFLUENCE OF ASCORBIC ACID ON ANAPHYLAXIS IN GUINEA PIGS. Sidney Raffel and R. R. Madison. Jour. Inf. Dis., lxiii (1938), p. 71.

Daily parenteral administration of ascorbic acid prior to and during the period of active sensitization caused complete inhibition of hypersensitive manifestations, anaphylaxis and uterine horn contraction in only two of 16 guinea pigs. The development of precipitins in these ascorbic acid-treated animals was marked as contrasted with untreated controls. The presence or absence of circulating antibody did not influence the hypersensitive reactions by an antianaphylactic or other effect. Single preshock injections of ascorbic acid into passively sensitized guinea pigs apparently had no effect on sensitivity. Ascorbic acid added to the suspension fluid in no way altered the sensitivity of excised uterine horns of passively sensitized animals.

A POSSIBLE MECHANISM OF LOWERED RESISTANCE TO PNEUMONIA. W. J. Nungester and R. G. Klepser. Jour. Inf. Dis., lxiii (1938), p. 94.

Mucin injected intrabronchially favored the production of pneumonia in rats sprayed one day after with pneumococci. Certain factors, as exposure to cold, prolonged deep ether anesthesia or alcoholic intoxication increased the aspiration of mucous material placed in the nose of white rats. Such factors also increased the incidence of pneumonia if pneumococci and mucin were previously inoculated intranasally. Cold or alcoholic intoxication was found to interfere with the closing of the glottis, thereby permitting the aspiration of mucin and pneumococci.

GONADOTROPIC ACTION OF NORMAL MALE URINE EXTRACT ON THE DOG. J. H. Leathem and J. A. Morrell. Endocrinol., xxiii (1938), p. 164.

An extract of normal male urine was found comparable to menopause urine extract in inducing estrus reactions in eleven of the twelve normal dogs tested. Other indications of the gonadotropic activity in the manner of a follicle stimulator were ascertained by excessive follicle stimulation, in which the ovary increased in weight to as much as eight times that of a normal anestrous ovary, the resorption of embryos in a pregnant animal, and the failure to affect a castrated dog. Normal male urine extract was found capable of inducing all the external manifestations of estrus; namely, vulval swelling, hemorrhagic discharge and estrus as determined by acceptance of the male. In addition, endometrial hypertrophy and vaginal smear changes also resulted. Some specific differences are cited in comparison with spontaneous estrus and estrus induced with menopause urine extract. Ovulation was determined by pregnancy in which one of five dogs tested became pregnant, delivered and suckled two normal pups. Injection of follutein on the first day of estrus caused ovulation in the one case tested.

ON THE CAUSE OF THE ANEMIA IN THE BARTONELLA INFECTION OF RATS. David Weinman. Jour. Inf. Dis., lxiii (1938), p. 1.

Bartonella muris anemia of rats is due to blood destruction and is accomplished by hemolysis. When *in vivo* hemolysis occurs, hemolysis *in vitro* can be demonstrated. Extra-corporeal hemolysis takes place with thrice washed erythrocytes in isotonic saline solution or with unwashed red cells in serum. It occurs both at 8° C. and at 37° C. and is more marked at the higher temperature. *In vitro* hemolysis is not due to a cause residing in the serum or plasma. The parasite appears to be the direct cause of erythrocytolysis and thus hemoglobin liberation, for (1) it is present within or attached to red cells when lysis occurs, (2) when no parasites are visible there is no demonstrable lysis, (3) fragmented red cells are frequently encountered and the cell remnants contain parasites and (4) the more fragile and achromic of the red cells are the parasitized ones.

THE EFFECT OF MANGANESE, ZINC, ALUMINUM AND IRON SALTS ON THE INCIDENCE OF PEROSIS IN CHICKS. W. M. Insko, Malcolm Lyons and J. Holmes Martin. Poultry Science, xvii (1938), p. 264.

These experiments confirm the work of previous investigators that manganese (35 parts per million in the form of manganese sulfate) has a protective action against slipped tendon when fed to growing chicks. However, they also show that no protective action occurs from aluminum and zinc as has been reported by other investigators. When 30 parts per million each of aluminum and zinc were used, no protective action was apparent but rather the incidence of slipped tendon was increased by the addition of these two elements to a ration containing 0.61 per cent calcium, 0.49 per cent phosphorus and 4.5 parts per million of manganese. Under the conditions of the experiments aluminum and

zinc were regarded as a causal factor rather than having a protective action.

A RETICULOCYTOGENIC AGENT FOR GUINEA PIGS PRESENT IN CERTAIN GASTRO-INTESTINAL SECRETIONS. J. Goodfriend, E. Chain and H. W. Florey. Quart. Jour. Exp. Physiol., xxviii (1938), p. 115.

A reticulocytogenic agent for reactive guinea pigs was found in the pyloric, duodenal and ileal secretions of the pig, in the pyloric and duodenal secretions of the cat and in the duodenal secretions of the rabbit and goat, but not in the fundic secretions of either pig or cat. The sites of the production of the agent correspond to those sections of the gastro-intestinal mucosa which have been found by Meulengracht to yield therapeutic agents active in pernicious anemia. From the duodenal secretions of the goat and the pig a substance has been prepared which is reticulocytogenic for guinea pigs in very small amounts when injected subcutaneously. The agent is thermolabile. It is soluble in acetone and in alcohol and is partially precipitated by mineral acids. It is dialysable through cellophane and collodion membranes. The molecular weight is therefore less than 4,000. It is not destroyed by pepsin or trypsin.

ON THE INHERITANCE OF PENDULOUS CROP IN TURKEYS (*Meleagris gallopavo*). V. S. Asmundson and W. R. Hinshaw. Poultry Science, xvii (1938), p. 276.

Pendulous crop is an inherited abnormality observed in turkeys raised where the maximum temperatures and hours of sunshine are relatively high and the relative humidity is relatively low. There were no cases of pendulous crop among the progeny of pendulous-crop birds when raised where the mean maximum air temperature was about 74° F. with fewer hours of sunshine and high humidity whereas 67.2 per cent of full brothers and sisters of these birds developed pendulous crops when raised where the mean maximum temperature was about 20° F. higher with more hours of sunshine and lower humidity during the summer growing periods. The tendency to develop pendulous crop under certain climatic conditions is determined by a recessive autosomal gene or genes.

AN IMPROVED CRITICAL TEST FOR POULTRY TAENIACIDES. Paul Harwood and Arthur C. Jerstad. Poultry Science, xvii (1938), p. 295.

In a series of tests for the removal of *Raillietina cesticillus* of chickens a mixture of drugs containing kamala was given to six chicks, each of which had been artificially infected with a single tapeworm. At autopsy one week after treatment each of five of the experimentally infected chicks was infested with a single species of *Raillietina cesticillus*. The fate of the single tapeworm in one chick could not be explained. The authors regard the method used as an improved critical test for poultry taeniacides.

THE EFFECT OF CASTRATE IMMUNE HORMONE ON THE TESTIS. H. S. Rubinstein. Endocrinol., xxiii (1938), p. 171.

Follicle stimulating hormone obtained from menopausal and castrate blood and urine induced premature testicular descent in normal miniature albino rats. It increases the weight of the testes significantly in both mature and immature animals although the effect is greater in the younger group. Interstitial tissue is increased in old and young rats but is greater in the latter. Tubular diameter is increased in the adult testis. Increased germinal cell proliferation results from the use of this hormone as noted from increase in mitotic forms in the testes and the finding of cells in the epididymis tubule when none are seen in the controls. The epididymis in this respect may serve as an index to testicular function. Although germinal cell proliferation is increased maturation is not hastened. Follicle stimulating hormone has no effect on general body growth.

Regular Army

The appointment of 1st Lieut. Stephen Grieve Asbill, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from September 8, 1938, and his assignment to station at the Presidio of Monterey, Calif., are announced. Lieutenant Asbill will proceed to the station to which assigned and report to the commanding officer for duty.

Lt. Colonel Howard M. Savage, V.C., having been found by an Army retiring board incapacitated for active service on account of disability incident thereto, and such findings having been approved by the President, the retirement of Lt. Colonel Savage from active service on September 30, 1938, under the provisions of section 1251, Revised Statutes, and the act of Congress approved April 23, 1930, is announced.

1st Lieut. Walter T. Carll, V.C., is relieved from assignment and duty at Fort Hoyle, Md., is assigned to the Army Veterinary School, Army Medical Center, Washington, D. C., and will proceed to that station and report to the commanding general, Army Medical Center, for the purpose of pursuing a course of instruction.

Lt. Colonel Jean R. Underwood, V.C., is relieved from his present assignment and duty at the Army Medical Center, Washington, D. C., effective at such time as will enable him to comply with this order, and will proceed to New York, New York, and sail on or about February 7, 1939, via Government transportation, for the Philippine Department. Upon arrival he will report to the commanding general for assignment to duty with the Veterinary Corps.

The promotion of 1st Lieut. Lloyd C. Tekse to the grade of captain with rank from September 30, 1938, is announced.

Veterinary Corps Reserve

New Acceptances

(First lieutenants)

Abraham, Max Carl, Edon, Ohio.

Allen, Jap Calvin, Route No. 2, Marshville, N. C.

Groves, Lawrence Wesley, 120 W. 9th St., North Platte, Nebr.

Todd, Wm. Charles Jr., 697 Pennington Ave., Trenton, N. J.

Separations

First Lieut. Stephen Grieve Asbill, accepted appointment in Regular Army. First Lieut. Richard Allen Goodman, died July 2, 1938.

Promotions

To Major: John Warden Burke, 337 Kenwood Ave., Dayton, Ohio.

To Captain: Alvin Rutti McDonald, 6 Marshall, Hartford, Conn.

To Captain: Arthur Bartlett Rogers, 1102 State Office Building, Richmond, Va.

To Captain: Herbert Franklin Sibert, Nelson, Nebr.

New Assignments to Active Duty with CCC

1st Lieut. Milton W. Firestone, Schenectady, N. Y. 1st Lieut. Aaron Francis Allison, Fort Oglethorpe, Ga. 1st Lieut. Russell James Stewart, Omaha, Nebr. 1st Lieut. James Howard Watson, Fort Leavenworth, Kans.

Termination of Assignment to Active Duty with CCC

1st Lieut. Harry Ruyle Lancaster, Fort Benning, Ga. Capt. Houston Odom, Fort Barrancas, Fla.

BUREAU TRANSFERS

Dr. Anthony J. Matter (O. S. U. '16), from Des Moines, Iowa, to Pierre, S. Dak., on tuberculosis eradication.

Dr. Irvin Myers (O. S. U. '13), from Cincinnati, Ohio, to Columbus, Ohio, on meat inspection.

Dr. Wm. F. Biles (K. C. V. C. '14), from Frankfort, Ky., to Jefferson City, Mo., in charge of tuberculosis eradication, etc.

Dr. Ralph Graham (Iowa '02), from Jefferson City, Mo., to Frankfort, Ky., in charge of tuberculosis eradication, etc.

Dr. Howard H. Cohenour (McK. '01), from Bismarck, N. Dak., to Salt Lake City, Utah, in charge of field investigations and tuberculosis eradication.

Dr. Harry E. Kemper (K. C. V. C. '14), from Albuquerque, N. Mex., to Bismarck, N. Dak., in charge of field investigations and tuberculosis eradication.

Dr. Bruce Kester (O. S. U. '25), from Cleveland, Ohio, to Hallstead, Pa., in charge of meat inspection.

Dr. Reuben C. Lambert (K. C. V. C. '17) from Pittston, Pa., to Wilmington, Del., in charge of meat inspection.

Dr. Oscar Nelson (U. P. '05), from Hallstead, Pa., to Jersey City, N. J., in charge of meat inspection.

Dr. Donald E. Cooperrider (O. S. U. '36), from Newark, N. J., to Cleveland, Ohio, on meat inspection.

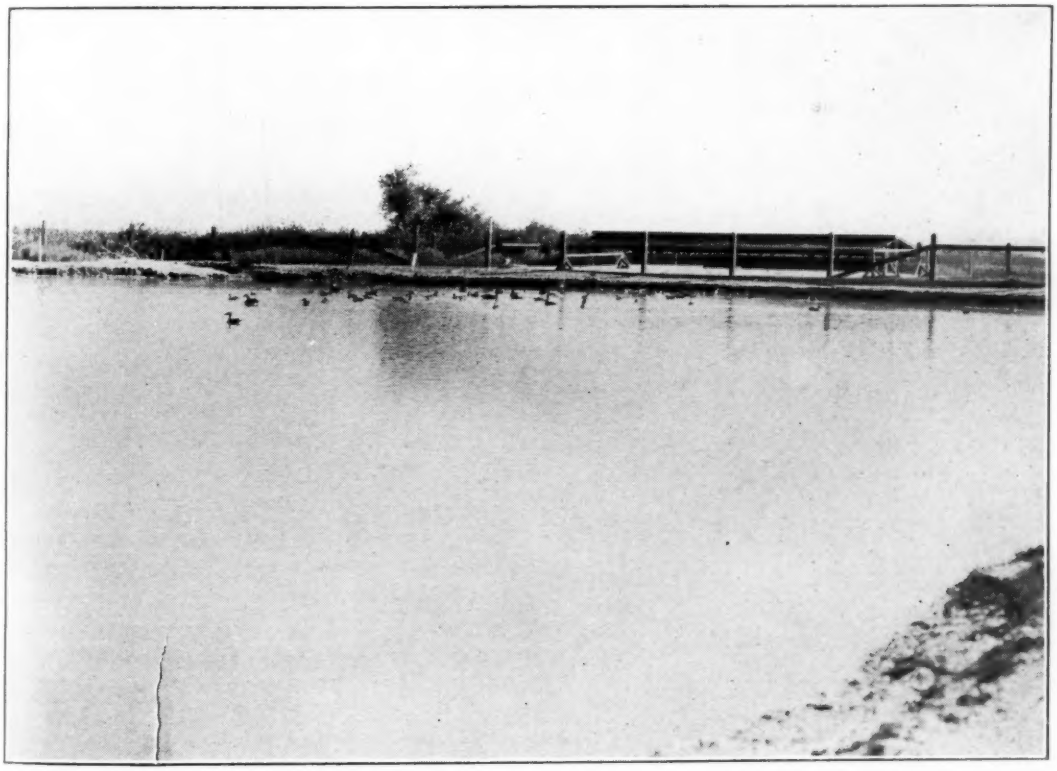
Dr. L. C. Heemstra (I. S. C. '32), from Jacksonville, Fla., to Agricultural Research Center, Beltsville, Md.

MISCELLANEOUS

Head of Regional Poultry Research Laboratory Appointed

Dr. J. Holmes Martin, professor of poultry husbandry and genetics at the University of Kentucky, has been appointed director of the new Regional Poultry Research Laboratory at East Lansing, Michigan, according to an announcement made recently by Dr. J. R. Mohler, chief of the U. S. Bureau of Animal Industry. Dr. Martin was at the University of Kentucky for 21 years, served for several years as editor of *Poultry Science*, and is the author of numerous scientific articles in the field of poultry husbandry.

In his position at East Lansing, Dr. Martin will direct the work of the new Poultry Research Laboratory and coördinate its research activities with those of the 25 agricultural experiment stations in the north central and northeastern sections of the United States, coöperating in the attack on problems affecting the viability of poultry, according to Dr. Mohler. Dr. Martin will continue his duties at the University of Kentucky until January 3, 1939.



HOLDING-PEN AND HOSPITAL FOR WILD WATERFOWL AFFECTED WITH BOTULISM AT THE BUREAU OF BIOLOGICAL SURVEY WILDLIFE DISEASE RESEARCH STATION, BRIGHAM, UTAH.

New Laboratory for Studying Diseases of Fur Animals

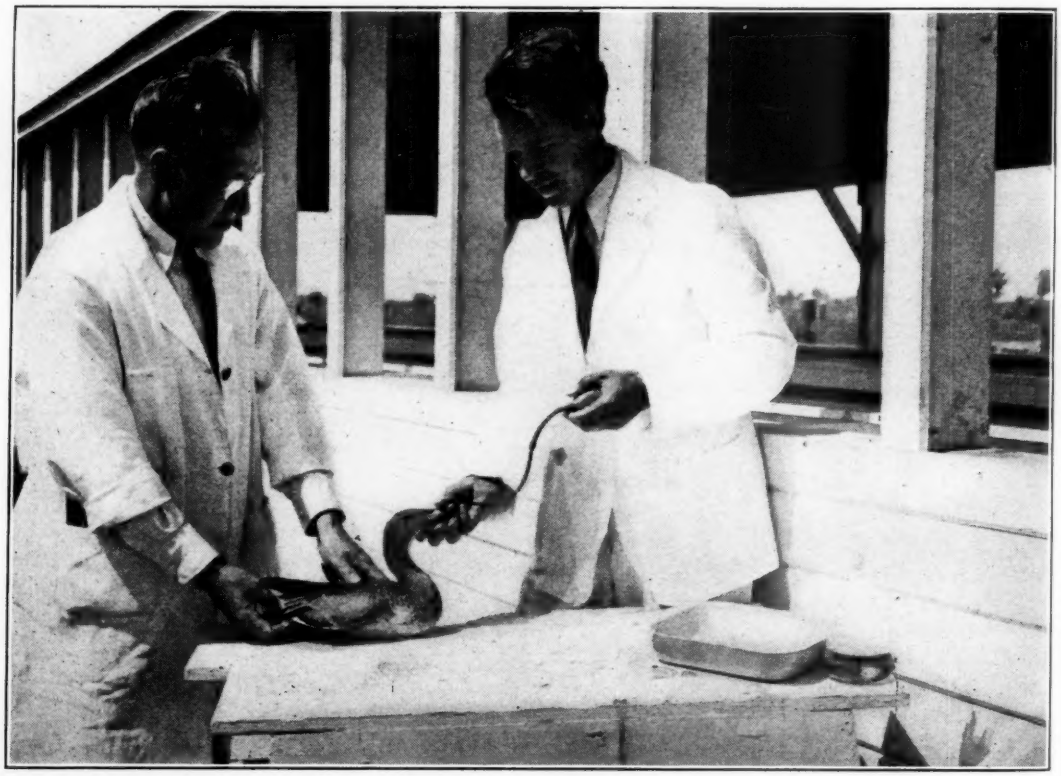
A branch laboratory for research in the diseases of fur animals is being established at Pullman, Wash., under a coöperative agreement between the State College of Washington and the U. S. Biological Survey. Dr. F. D. McKenney, of the Section of Disease Control of the Bureau, will be in charge of the laboratory. Arrangements for maintaining test animals at the college and facilities for disease investigations are being completed.

Dr. J. E. Shillinger, in charge of the Section of Disease Control, has also announced the transfer of Dr. Don R. Coburn from the Bear River Migratory Bird Refuge, Utah, and the proposed addition of an assistant veterinarian to the new pathological laboratory of the Bureau at the Patuxent Research Refuge, near Bowie, Md. Doctor Coburn's disease investigations at the Bear River Refuge will be continued by Dr. E. R. Quortrup.

"Stationing members of the disease control staff in various parts of the country where facilities for pathological research have been provided," says Dr. Shillinger, "will help the Survey in clearing up some of the questions on infectious, parasitic and nutritional diseases, so that methods of treatment or control may be improved."

Chappel Foundation Opens Nominations for Its 1939 Award

Nominations for the Chappel Award, to be presented by the Chappel Foundation, of Rockford, Illinois, to the person who has contributed the most distinguished service to American dogdom during 1938, opened on October 10, according to Mr. Walter E. Armstrong, Director of the Foundation. Applications, he disclosed,



DR. DON R. COBURN (LEFT) AND DR. E. R. QUORTRUP (RIGHT) GIVING EXPERIMENTAL TREATMENT TO WILD DUCK AFFECTED WITH BOTULISM AT BUREAU OF BIOLOGICAL SURVEY WILDLIFE DISEASE RESEARCH STATION, BRIGHAM, UTAH.

will be accepted up to and including December 15. This marks the fifth consecutive year that the highly-prized bronze plaque has been offered.

As in previous years, anyone may make a nomination, and anyone who has broadly distinguished himself by contributing to the welfare of American dogdom may be nominated. Mr. Armstrong added that the names of the judges and the date of the actual presentation will be disclosed later. Past winners have been Mr. George F. Foley, Mr. James W. Spring, Mrs. M. Hartley Dodge and Mrs. George Bethune Adams. Who will be chosen this year? Address your nominations to Chappel Kennel Foundation, Rockford, Illinois.

WPA Publishes Summary and Index of Research

The results of some 2,000 research projects, carried on as part of the federal work relief program, are summarized briefly in a digest and index which has been published by the Works Progress Administration. This volume of 291 pages contains a concise statement of the principal conclusions of each study and an alphabetical subject index to the contents.

The reports on these projects touch upon nearly every field of natural and social science and many of them have appeared in the form of articles in scholarly journals. However, several hundred of the reports summarized in this index are in manuscript form, and arrangements have been made with the American Documentation Institute whereby microfilm copies of the original reports will be furnished at nominal rates for the use of research specialists.

A small edition of this volume has been prepared for distribution to the larger public and university libraries, where it will be available for reference, and for government departments, industrial concerns and research foundations. A limited supply of copies of this Index of Research Projects is still available. Requests should be addressed to the Works Progress Administration, Washington, D. C.

SURVEY ON EQUINE ENCEPHALOMYELITIS

The following report has been compiled from replies received to a letter sent to all state live stock sanitary control officials, under date of August 20, asking for information concerning the prevalence of equine encephalomyelitis in each state. Replies were received to 29 letters, giving information on the prevalence or non-existence of the disease at that time (latter part of August). Later reports were received from Colorado, Indiana, Iowa, Minnesota, Nevada and Tennessee, and this later information is included in the reports for these states.

Alabama: No cases diagnosed positively.
I. S. McADORY,
State Veterinarian.

Arizona: No report.

Arkansas: About 15 cases reported during August.
C. D. STUBBS,
State Veterinarian.

California: No report.

Colorado: Disease appeared first in June, three weeks earlier than usual. Almost every county in state infected. Disease practically abated by September 10.
R. M. Gow,
Livestock Sanitary Commissioner.

Connecticut: One case reported up to August 24. Vaccination being practiced extensively. Horses being removed from pasture, especially at night.
GEO. E. CORWIN,
Deputy Commissioner on Domestic Animals.

Delaware: No report.

Florida: No report.

Georgia: No report.

Idaho: No report.

Illinois: No report.

Indiana: No cases reported up to August 1. From August 10 to 25, suspected cases reported from seven counties. By October 1, practically the entire state covered with isolated cases. Disease much more prevalent in some localities than in others.
J. L. AXBY,
State Veterinarian.

Iowa: Reports from 355 veterinarians show about 25,000 cases, with a death loss of about 5,000 head, up to September 9. All counties in state infected.
H. A. SEIDELL, *Chief,*
Division of Animal Industry.

Kansas: Considerable number of cases all over the state. Most prevalent in the southeast part.

WILL J. MILLER,
Live Stock Sanitary Commissioner.

Kentucky: No cases reported up to August 25.

D. E. WESTMORLAND,
State Veterinarian.

Louisiana: Three cases in June, all fatal. Several other cases later, in ten parishes. No cases reported since August 15.

E. P. FLOWER,
Secretary and Executive Officer.

Maine: No report .

Maryland: No report.

Massachusetts: Disease appeared first about August 15, in towns along the Rhode Island line, Rehoboth, Dighton and Taunton. It then spread through the Bridgewaters, Middleboro, Brockton, Stoughton and Easton.

CHARLES F. RIORDAN, *Director,*
Division of Livestock Disease Control.

Michigan: No report.

Minnesota: Under date of October 7, there had been 4,909 reports received, covering 5,838 infected horses, with 1,225 fatalities.

CHAS. E. COTTON,
Secretary and Executive Officer.

Mississippi: No cases reported up to August 30.

E. S. BRASHIER,
State Veterinarian.

Missouri: Some cases reported but no authentic information available under date of August 25.

H. E. CURRY,
State Veterinarian.

Montana: Incomplete reports, received up to August 24, indicated that there had been 1,088 cases in 43 counties, with deaths of 170 horses.

H. F. WILKINS,
Acting State Veterinarian.

Nebraska: No report.

Nevada: No cases up to July 1. Nine cases reported in July, 23 during August, and 11 in September. Immunization extensively practiced.

WARREN B. EARL, *Director,*
Division of Animal Industry.

New Hampshire: No cases reported up to August 25.

R. W. SMITH,
State Veterinarian.

New Jersey: One case, in Salem County, up to August 25. Suspected cases in two other counties. Many horses in southern part of state immunized.

R. A. HENDERSHOTT, *Chief,*
Bureau of Animal Industry.

New Mexico: No report.

New York: No cases reported up to August 26.

E. T. FAULDER, *Director,*
Bureau of Animal Industry.

North Carolina: Disease reported from 13 counties up to September 2.

WM. MOORE,
State Veterinarian.

North Dakota: Disease appeared the latter part of June. From 80 to 90 per cent of the affected horses recovered. Information on number of cases not available.

T. O. BRANDENBERG,
State Veterinarian.

Ohio: No report.

Oklahoma: Up to August 23, there were reports of 148 cases, with 32 deaths, in 33 counties. The first case was reported from Cherokee County, in March.

D. H. RICKS,
State Veterinarian.

Oregon: No report.

Pennsylvania: No cases reported up to August 25.

H. M. KALODNER, *Director,*
Bureau of Animal Industry.

Rhode Island: Disease appeared in Providence County about August 1. Second case appeared about 12 days later, near the Narragansett race track. Third case on August 17. Four days later, disease appeared in Bristol County. All cases were fatal. Much vaccinating being done.

JOSEPH S. BARBER,
State Veterinarian.

South Carolina: No report.

South Dakota: No report.

Tennessee: No cases reported up to September 30.

A. C. TOPMILLER,
State Veterinarian.

Texas: Disease appeared during latter part of July, in the northwestern section of the state. Disease appeared to be more virulent this year than before. Mortality expected to be above 50 per cent.

T. O. BOOTH,
State Veterinarian.

Utah: No report.

Vermont: One case diagnosed clinically on August 14. Several other suspected cases reported.

R. S. GOING, *Chief,*
Live Stock Division.

Virginia: No report.

Washington: In Franklin and Benton counties, some 30 head of horses died from the disease.

M. R. HALES, *Supervisor,*
Dairy and Live Stock Division.

West Virginia: No cases reported up to August 25.

H. M. NEWTON,
B. A. I. Inspector in Charge.

Wisconsin: No report.

Wyoming: First case reported on July 5. Seventeen counties infected up to August 26, with 1,623 cases and 377 deaths.

H. D. PORT,
State Veterinarian.

New Veterinary Clinic Building for Michigan State College

On October 21, the Governing Board of Michigan State College awarded the contract for the construction of an addition to the Veterinary Clinic Building on the East Lansing campus. The accompanying photograph shows the architect's conception of the proposed addition.

The cost of the structure will be $133,000 and it is estimated that the accommodations for the small-animal clinic will be practically doubled. In addition, the new building will house the Physiology and Pharmacology Department and it will contain an auditorium with a seating capacity of approximately 300 people.

NEW VETERINARY CLINIC BUILDING AT EAST LANSING

The present Veterinary Clinic Building has available about 15,000 square feet of floor space. About 26,000 square feet will be provided in the addition covered by the contract. The addition in the rear would provide about 7,000 square feet.

Eastern Iowa Silver Jubilee

The annual meeting of the Eastern Iowa Veterinary Association, held at Cedar Rapids, October 18-19, 1938, took the form of a Silver Jubilee celebration. The fact that 268 veterinarians and 141 ladies were in attendance is the best indication of the success of the meeting.

Veterinarians of national prominence contributed to the Twenty-fifth Anniversary Technical Program. These included: Dr. William H. Feldman, Mayo Foundation, Rochester, Minn.; Dr. H. D. Bergman, president of the American Veterinary Medical Association; Prof. W. E. Petersen, University of Minnesota; Dr. A. T. Kinsley, Kansas City, Mo.; Dr. Frank Breed, Lincoln, Neb.; Dr. George R. Fowler, Iowa State College; Dr. C. L. McGinnis, Peoria, Ill.; Dr. A. F. Schalk, Ohio State University; Dr. C. L. Campbell, Saint Louis, Mo.; Dr. Ashe Lockhart, Kansas City, Mo., and Dr. L. A. Merillat, Chicago, Ill.

The annual banquet and ball attracted more than 300 persons. A special program had been arranged by a committee headed by Dr. Fred J. Crow, of Iowa City. Dr. A. R. Menary, of Cedar Rapids, officiated as toastmaster. Dr. Bergman was the principal speaker. In his address he traced the history of the Eastern Iowa Veterinary Association and referred to the organization as a pioneer in its particular field of service.

WANTED

The accompanying photographs are of Joseph Paul Cretzer and Edna May Cretzer, husband and wife. These individuals have used numerous aliases other than their correct names and federal processes are outstanding for their arrest, Joseph Paul Cretzer being wanted particularly for having engaged in a series of bank robberies on the Pacific Coast.

WANTED

Joseph Paul Cretzer (left): Age, 28; height, 5 ft. 7 in.; weight, 170 lbs.; build, medium heavy; hair, dark brown or black; complexion, olive.

Edna May Cretzer (right): Age 30; height, 5 ft. 1 in.; weight, 102 lbs.; eyes, brown; hair, auburn (dyes it various colors); complexion, fair; build, small.

These individuals have in their possession a registered male Pekingese known as "Sir See-Wee." This dog is also referred to as "Squeegie," "Skeezix," and possibly "Fo-Fo." This dog is slightly over two years of age and in all respects is reported to be a good specimen of the Pekingese breed.

It has been the practice of these individuals to take this dog to a veterinarian upon the slightest provocation and, accordingly, it is hoped that these fugitives may be located thereby. Any information you are able to furnish will be sincerely appreciated and will be maintained as confidential. The same should be forwarded to the Director, Federal Bureau of Investigation, United States Department of Justice, Washington, D. C.

NEW YORK WORLD'S FAIR EXHIBIT

On May 17, 1938, the following letter was sent to the secretaries of all veterinary organizations in the United States:

You will recall that last June, Dr. H. Preston Hoskins, Secretary of the American Veterinary Medical Association, communicated with you relative to tentative plans recommended by the Executive Board of that association for two veterinary exhibits, one to be placed at the New York World's Fair and the other at the San Francisco exhibition during 1939.

It has since developed that these plans were too ambitious and it was found that with the funds which would probably be available it would not be possible to put on a credible exhibit at both fairs. Accordingly, as Dr. Hoskins has advised you, these plans had to be given up and the contributions returned because they were collected with the understanding that two exhibits would be prepared. Notwithstanding this, the Executive Board of the A. V. M. A. has appointed me as Chairman of the New York World's Fair Committee and it is my opinion that we should not completely give up this unusual opportunity to publicize our profession. I am therefore suggesting that we revise our plans and concentrate our efforts in making possible a satisfactory exhibit at the New York World's Fair.

All preliminary estimates and predictions indicate that this will be one of the largest and best fairs ever held and I feel that we should not miss this opportunity to publicize veterinary activities by placing an instructive exhibit where it can be seen by the 15 millions of people who are expected to visit this fair. The proposed exhibit will be general in scope. It will not feature any particular branch of the profession over another, but will be one that will emphasize to the country the importance of the service veterinarians as a class are rendering. No promise can be made at this time as to the ultimate disposition of the exhibit after the fair is over.

We have been tentatively allotted 400 square feet of space at $50 a square foot or $20,000. Of this amount $14 per square foot or $5,600 is for rental, the balance being for cost of building the exhibit, lighting, guards, janitors, and other overhead charges made by the fair organization for the six months' period. I have received promises of $11,000 from persons not directly interested in our activities, so it remains for those veterinarians who wish to see our profession in line with medicine, dentistry, engineering, etc., to help make up the balance in order for this undertaking to be successful. The other professions mentioned will be represented by considerably more space, and those of us who have studied the proposal feel that 400 square feet is the least amount in which a creditable veterinary display can be made.

As this idea originated, as far as I know, in the A. V. M. A., I have asked the Chairman of the Executive Board to see that $5,000, or

as much thereof as is needed, be made available from the treasury of the A. V. M. A. This leaves $4,000 to be contributed by associations such as yours, and I will appreciate it if you will take this matter up with your organization at its next meeting, advising me of whatever action is taken. One-half of your subscription may be paid this year and the balance early in 1939. Any amounts subscribed will be held in a special fund which will be used for no other purpose.

As a result of the above letter I have received the following donations:

Central New York Veterinary Medical Association	$ 50.00
Connecticut Veterinary Medical Association	100.00
Hudson Valley Veterinary Medical Association	50.00
Kentucky Veterinary Medical Association	50.00
Massachusetts Veterinary Association	75.00
Veterinary Medical Association of New Jersey	75.00
Veterinary Medical Association of New York City	75.00
North Dakota Veterinary Association	12.50
Rhode Island Veterinary Medical Association	50.00
State Veterinary Medical Association of Texas	35.00
Washington State Veterinary Medical Association	25.00
Wyoming Veterinary Medical Association	20.00
E. R. Teich, B. A. I. Inspector, Velva, N. Dak.	5.00

In order that our profession may have as creditable an exhibition as those of the dentists, druggists, and physicians adjoining our space, I hope that the above donations will be promptly augmented by many others, as we must all recognize that the veterinarians of the United States are on trial and should not expect our friends to carry all the responsibilities of publicizing our activities.

JOHN R. MOHLER, *Chairman,*
Special Committee on World's Fair Exhibit.

Southern States Meeting

An unusually well-balanced program has been arranged for the 23rd annual meeting of the Southern States Veterinary Medical Association, to be held in Knoxville, Tenn., November 3-5, according to an advance announcement made by Dr. L. A. Mosher, Secretary. The papers will deal with diseases of horses and mules, cattle, swine, dogs and cats. Contributors to the program are listed from twelve states. Dr. Cassius Way, president-elect of the A. V. M. A., will contribute to both the literary program and the clinic. The third day of the meeting will be devoted to the clinic, which will be held in Temple Hall, University of Tennessee. Entertainment for the ladies has been arranged, concluding with a shopping tour on Saturday morning. The banquet will be held at the Andrew Johnson Hotel on Friday evening.

Concerning Eligibility for Attendance at the Conference of Official Research Workers in Animal Diseases of North America

The next annual meeting of the Conference of Official Research Workers in Animal Diseases of North America will be held at Hotel Sherman, Chicago, Tuesday, November 29. In order that eligibility for attendance at this meeting may be clearly understood, it seems advisable to quote Article II of the Constitution and By-laws of the organization. Article II reads:

> Research workers employed by the United States government or by any national, state or provincial government in North America or by endowed institutions and engaged in the investigation of animal diseases shall be eligible for active membership in the Conference.

WILLIAM H. FELDMAN, *Secretary-Treasurer.*

New Veterinary Building at Colorado State

The Colorado State Board of Agriculture has approved a request to the WPA for a new veterinary building at Fort Collins, to cost $127,000, 55 per cent of which is to be paid from state funds and 45 per cent from federal funds, according to an announcement made by Dean Newsom. Should this request be approved in Washington, it will give Colorado State College a much needed office and laboratory structure.

Errata

(September, 1938, JOURNAL)

Page 159. Col. 1, par. 1, line 3, and par. 2, lines 3 and 7, under "New Principal," "Oliver" should be "Olver."

Page 161. The name of the senior author is McKenney, instead of McKennedy.

Page 165. Footnote 2, line 5, "Gross" should be "Cross."

Page 165. Col. 2, par. 1, line 6, "Bekefeld" should be "Berkefeld."

Page 166. Table I, line 1, "Overheating" should be "Overeating."

Page 166. Col. 2, par. 5, line 2, "of" should be "to."

Page 167. Col. 1, par. 2, line 10, "370° C." should be "37° C."

Page 167. Reference 4, "Oxter" should be "Oxer."

Page 208. The new location of Dr. Willard J. Barga (O. S. U. '38) was incorrectly given as Dayton, Ohio. He is located at Versailles, Ohio.

Page 208. The home address of Dr. L. C. Shaw is Geneva, Neb., instead of Staceyville, Iowa. The latter is the address of Dr. B. T. Hartnell.

Page 208. Dr. Stephen H. Swain, of Decatur, Ill. is a non-graduate and not a graduate of McKillip Veterinary College, 1909.

Page 208. Dr. C. F. Runnels is located at Saint Clairsville, Ohio, and not Clairsville.

Page 208. Dr. W. G. Brock is a graduate of Ohio State University, 1911. He was reported as a graduate of the Ohio Veterinary College.

(October, 1938, JOURNAL)

Page 226. Fig. 1. Legend should read, "Encephalomyelitis, sleepy type, showing animal just prior to falling."

Page 228. Col. 2, par. 2, line 10, "strain" should read "strains."

Page 229. Fig. 2. Legend should read, "Snarling symptom often seen in connection with the second epizoötic." (This illustration does not show lip paralysis or pendulous type of true equine encephalomyelitis and would warrant severe criticism if left uncorrected.)

Page 230. Fig. 3. Legend should read, "Equine encephalomyelitis, typical sleepy posture." (Neither this nor any other horse affected with true equine encephalomyelitis was observed to eat dirt, although this symptom was commonly seen during the second epizoötic, as indicated in the text.)

Page 230. Col. 2, par. 2, line 4, "Hadley" should be "Hadleigh." Same error appears on p. 231, col. 1, par. 2, line 2.

Page 231. Col. 1, par. 1, line 2, "aerobices" should be "aerobes."

Page 232. References 9 and 10 should be combined to read, "Thorp, Frank, Jr.: Personal communication. Data compiled from reports of veterinary practitioners by E. N. Stout." Reference 11 should be 10, and reference 12 should be 11.

Page 236. Date of receipt for publication should be June 9, 1938, instead of January 13, 1937.

Page 239. Figure 1 belongs to article, "Pyelonephritis in Cattle," by C. C. Palmer, pp. 241-243.

Page 243. Col. 1, par. 2, line 11, under "The Soybean," Illinois should be "Indiana."

Page 251. Col. 1, par. 1, line 2, "Lienhart" should be "Lienhardt."

Page 254. Col. 2, head should read "American Chemical Society," instead of "Animal Chemical Society."

Page 255. Date of receipt for publication should be August 22, 1938, instead of August 22, 1937.

Page 278. The college and year of graduation for Dr. W. L. Drinkwater should have been Ontario, 1886, instead of U. S. C. V. C., 1919.

Drug Manufacturer Fined

In Federal Court in Kansas City, Mo., recently, Samuel E. Massengill, head of the Massengill Manufacturing Company, which sold the elixir of sulfanilamide that was responsible for the deaths of a number of persons about a year ago, pleaded guilty and was fined $9,300. The government charged that 73 deaths were directly attributable to the use of this drug.

NECROLOGY

THOMAS ORLA KELLER

Dr. Thomas O. Keller, of Peru, Ind., died at his home on August 8, 1938, following an illness of four months, due to heart trouble and complications.

Born at Converse, Ind., on May 5, 1883, Dr. Keller was graduated from the McKillip Veterinary College in 1912. He practiced first at Ridgeville, Ind., before going to Peru. He served as mayor of Peru from 1926 to 1930, the first Republican to be elected to that office in 40 years.

Dr. Keller joined the A. V. M. A. in 1915. He held a commission in the Veterinary Reserve Corps and was a member of the Masonic Lodge of Andrews, Ind. He is survived by his widow (née Daphne Landers) and two brothers.

FRED H. GULDAGER

Dr. Fred H. Guldager, of San Francisco, Calif., died suddenly on August 17, 1938. His death was due to a heart attack.

Born in Neenah, Wis., October 16, 1879, Dr. Guldager served with the 35th Infantry, U. S. Volunteers, in the Philippines, during the Spanish-American War. He was also a veteran of the World War, having been commissioned as a second lieutenant and stationed at Camp Greenleaf, Ga.

Dr. Guldager was a graduate of the San Francisco Veterinary College, class of 1911. Immediately following graduation, he entered state service and remained in this work almost continuously. He took an active part in the long campaign of sheep scab eradication in California, which was brought to a successful conclusion in 1930. During the past several years, he was stationed in San Francisco, engaged in stockyards, port and poultry inspection work. He is survived by his widow. C. U. D.

DAVID SCOTT KAY

Dr. David S. Kay, of Los Angeles, Calif., died suddenly, at his home, on September 4, 1938. He had just returned from a pleasure outing with his family. Death was due to cardiac myositis.

Born in Fifeshire, Scotland, October 2, 1883, Dr. Kay came to the United States about 1898. He was a graduate of the San Francisco Veterinary College, class of 1911. Later, he received a Bachelor of Science degree from the University of Nevada and a master's degree in Animal Husbandry from the University of California.

Both prior to and following the World War. Dr. Kay engaged in practice in California. During the war he served in the A. E. F. and was decorated with the Croix de Guerre. About 1925, he entered the service of the U. S. Bureau of Animal Industry and continued in this work until his death.

Dr. Kay joined the A. V. M. A. in 1928. He was a member of the National Association of B. A. I. Veterinarians, the American Legion, and Union Lodge No. 58, F. and A. M., of Sacramento. He is survived by his widow (née Janet Ramsay) and three daughters. C. F. K.

ALBERT T. PETERS

Dr. Albert T. Peters, of Peoria, Ill., died at his home on September 11, 1938, after an illness of several months.

Born in Chicago, on February 10, 1868, Dr. Peters studied veterinary medicine at the University of Stuttgart, Germany. He returned to the United States and accepted a position as pathologist at the University of Nebraska. He remained there for over 15 years and devoted much time to the study of the etiology of hog cholera. He

contributed numerous articles to the veterinary literature of this period.

In 1909, Dr. Peters returned to Illinois for the purpose of establishing the State Biological Laboratory at Springfield. This laboratory produced anti-hog cholera serum and virus on a large scale. Three years later, he was appointed Chief Veterinarian of the State Department of Agriculture. He remained in this position until 1920, when he resigned to enter the commercial field. He was one of the organizers of the Vitamineral Products Company, with which he was identified at the time of his death.

Dr. Peters joined the A. V. M. A. in 1893, but resigned in 1920, when he entered commercial work. He was secretary of the Illinois Breeders Association, a member of the Board of Directors of the Peoria Y. M. C. A., a member of the Peoria Rotary Club and former Governor of Rotary International.

As head of the Crippled Children's Committee of the Peoria Shrine, Dr. Peters rendered outstanding public service. He was the first president of the Big Brothers and was also first president of the Big Brothers and Big Sisters, remaining at the head of the larger organization until shortly before his death.

Surviving are his widow (née Emma Rickert) and two daughters. Pallbearers were six past presidents of the Peoria Rotary Club.

OTIS M. GOODALE

Dr. O. M. Goodale, of Kewanee, Ill., died in the Kewanee Public Hospital, on September 27, 1938, following injuries received in an automobile accident the previous week.

Born in Toulon, Ill., April 16, 1870, Dr. Goodale attended Stark County schools, Toulon Academy and the Chicago Veterinary College. Following his graduation from the latter in 1893, he located in Princeville, Ill. In 1904, he removed to Kewanee and had practiced there ever since.

Dr. Goodale was a member of the Illinois State Veterinary Medical Association. He is survived by his widow (née Minnye C. Ahart), one son and one daughter.

ADAM ELGAS

Dr. Adam Elgas, of Hartford, Mich., died in Mercy Hospital, at Benton Harbor, Mich., on September 23, 1938, after an illness of less than a week. Death was ascribed to intestinal paralysis.

Born in Bainbridge Township, Berrien County, in 1859, Dr. Elgas was graduated from the Ontario Veterinary College in 1891 and practiced at Hartford, Mich., for 47 years. He had been one of the officials of the Van Buren County Fair since its organization in 1913. He is survived by his widow and two brothers.

LESTER WARREN THIELE

Dr. Lester W. Thiele, of Buchanan, Mich., committed suicide in his room at the Hotel Rex, that city, on September 22, 1938.

Born in Chicago, Ill., March 8, 1894, Dr. Thiele was graduated from the Chicago Veterinary College in 1914. During recent years he had practiced at various places in Wisconsin and Michigan, and had also served in the U. S. Bureau of Animal Industry on various assignments.

Death was ascribed to a self-administered dose of chloral hydrate, taken in a moment of despondency over family troubles.

JOHN DAWSON ADAMS

Dr. John D. Adams, of Boise, Idaho, died on September 23, 1938. He was engaged in private practice at the time of his death.

Born near Carthage, Ill., on September 4, 1873, he moved to Kansas, with his family, attended public schools and then took a course in the Central Business College, of Denver, Colo. He taught school for a year and, in 1900, entered the service of the U. S. Bureau of Animal Industry. He resigned in 1905 and entered Washington State College for the study of veterinary medicine.

Following his graduation in 1910, Dr. Adams practiced at Genesee and Moscow, Idaho. In 1916, he was appointed a member of the Idaho State Board of Veterinary Medical Examiners and served as treasurer of the Board. In 1919, he was appointed State Veterinarian of Idaho. He was com-

missioned as captain in the Veterinary Reserve Corps and was on active duty from 1934 to 1937 in connection with CCC work.

Dr. Adams joined the A. V. M. A. in 1915. He was a member of the Committee on Necrology, 1922-23. He was a member of the Idaho State Veterinary Medical Association, the Boise Rotary Club, and various Masonic bodies, including the El Korah Temple Shrine of Boise. He is survived by his widow (née Gladys Newsome) and two brothers. A. J. C.

ANTON HENRY HORN

Dr. Anton H. Horn, of Warrensburg, Ill., died in the Decatur and Macon County Hospital, on September 28, 1938, after an illness of only two days. He had overexerted himself in treating cases of equine encephalomyelitis in his territory.

Born near Evansville, Ind., January 18, 1897, Dr. Horn was graduated from the Saint Joseph Veterinary College in 1911. He had practiced at Warrensburg for about 15 years. He is survived by his widow (née Mary J. Woodside), two sons, two daughters, his father, three brothers and a sister.

CHARLES S. CHASE

Dr. Charles S. Chase, of Bay Shore, L. I., N. Y., passed away at his home on September 30, 1938, after a long and painful illness.

Born in Greenwich Village, New York City, on February 23, 1868, Dr. Chase entered the employ of Tiffany and Company at the age of twelve. After 24 years with this firm, he resigned to take up the study of veterinary medicine. In the meantime he had attended the College of Saint Francis Xavier and Cooper Union. He was graduated from New York-American Veterinary College in 1906 and immediately entered general practice at Bay Shore.

Dr. Chase served in the Veterinary Corps of the Army during the World War. He was commissioned as a second lieutenant on August 8, 1917; served on Purchasing Board for Public Animals, Eastern Purchasing Zone; post veterinarian at Fort Myer, Va.; instructor in the Veterinary Corps at Camp Greenleaf; promoted to first lieutenant, June 21, 1918; camp veterinarian at Camp Cody, Deming, N. Mex.; made captain, August 10, 1918; Division Veterinarian, 97th Division, September 19, 1918; honorably discharged, December 17, 1918.

Dr. Chase joined the A. V. M. A. in 1912 and served as Resident Secretary for New York, 1917-18. He was a member of the Veterinary Medical Association of New York City and the Long Island Veterinary Society. He was active in the American Legion, having held local, county and state offices. He was a member of the Twelfth International Veterinary Congress, the Bay Shore Rotary Club, the Masonic order, the Odd Fellows and the Knights of Pythias.

Among the survivors are his widow (née Martha Hulse), one son and two daughters. C. P. Z.

JOHN W. DOLD

Dr. John W. Dold, of Cincinnati, Ohio, died at his home on October 1, 1938, after a brief illness. Death was the result of a cerebral hemorrhage. He was 68 years of age. He was a graduate of the Cincinnati Veterinary College, class of 1920, and practiced in Northside up to the time of his fatal illness. He is survived by his widow, one daughter and three sons.

CLIFFORD P. MURRAY

Dr. Clifford Philemon Murray, of Glens Falls, N. Y., passed away at the Glens Falls Hospital, on October 6, 1938. He was accidentally shot in the abdomen with a .22 calibre pistol, while engaged in target practice with some friends, at his hospital one mile out of Glens Falls.

The deceased was a son of the late Dr. H. Clifford Murray (Ont. '02, N. Y.-Amer. '05), who died February 6, 1935. "Phil," as he was familiarly known, was graduated from the New York State Veterinary College at Cornell University in 1934. He was associated with his father until the latter's death. He recently built a hospital containing 42 kennels, an isolation ward with 15 kennels, and accommodations for 20 cats.

GLEANINGS FROM OUR CORRESPONDENCE

MARRIAGES

DR. ADOLF J. SYPIEN (M. S. C. '36), of Chicago, Ill., to Miss Anne Hacus, at Chicago, September 3, 1938.

DR. CHARLES K. JOSSE (O. S. U. '36), of Saint Louis, Mo., to Miss Ruth Ullery, of Covington, Ky., at West Milton, Ohio, September 28, 1938.

DR. CARL RALPH BENTON (Mich. '36), of Salem, Mass., to Miss Barbara Mildred Allen, of Concord, Mass., at Concord, October 2, 1938.

DR. I. M. JACKSON (Colo. '38), of Salt Lake City, Utah, to Miss Alice Patridge, of Windsor, Colo., September 4, 1938.

PERSONALS

DR. A. A. TURNER (Ind. '23) has taken over the practice of Dr. Roy E. Kluck, at Freeport, Ill.

DR. HORST SCHRECK (O. S. U. '17) has opened a new hospital at 2103 Texas Street, El Paso, Texas.

DR. J. J. ARNOLD (Mich. '37) addressed the Newcastle (Ind.) Kiwanis Club, the evening of September 27, at the Westwood Country Club.

DR. J. R. LUCAS (McK. '19), of Farmersville, Ill., suffered a broken leg while treating a horse affected with encephalomyelitis, on September 10.

DR. S. F. GAYNOR (Ind. '20), of Logansport, Ind., was knocked down and bitten by an angry sow on his farm near Fulton, Ind., in September.

DR. CHARLES M. CHASE (Colo. '19), of Indianapolis, Ind., was retired from service with the U. S. Bureau of Animal Industry, on May 31, 1938.

DR. T. P. POLK (U. S. C. V. S. '10), extension veterinarian of the University of Kentucky, has been confined to his bed with heart trouble, since September 28.

DR. MANLEY B. MATHERS (Chi. '15), of Mason City, Ill., was seriously injured on October 10, when he was gored by a bull elk on his stock farm near the city.

DR. WILLIAM F. OSBORN (K. C. V. C. '08) retired from the service of the U. S. Bureau of Animal Industry on July 31, 1938. He was stationed at Newark, N. J.

DR. WILLIAM C. BELLIS (Wash. '35), who is on meat inspection work for the California Department of Agriculture, has been transferred from Fall River Mills to Los Angeles.

DR. C. D. EBERTZ (Corn. '35) has entered private practice at Auburn, N. Y. He was formerly connected with the Department of Surgery and Medicine of Kansas State College.

DR. FONSOE M. BOLIN (Iowa '29) has reported a change of address from Oregon State College to the Department of Animal Pathology and Hygiene, North Dakota State College, Fargo.

DR. FRANK E. MURRAY (O. S. U. '92), who had been in charge of field investigations and tuberculosis eradication in Utah for the U. S. Bureau of Animal Industry for a number of years, was retired from active service on June 30, 1938.

DR. G. W. CHAPMAN (Chi. '08), of Webster, S. D., was operated on for acute appendicitis on October 2. The latest report is to the effect that he is making a very satisfactory recovery.

DR. J. G. CATLETT (U. S. C. V. S. '16), who spent the summer in New York as official veterinarian to the State Racing Commission, has returned to his home in Miami, Fla., for the winter.

DR. GEORGE W. GILLIE (O. S. U. '07), of Fort Wayne, Ind., has been waging a strenuous campaign for election to Congress. He is the Republican candidate from Fourth Congressional District in Indiana.

DR. LEONARD J. GOSS (O. S. U. '34), who was at the New York State Veterinary College, Cornell University, for several years, is now at the Kentucky Agricultural Experiment Station, Lexington, as assistant pathologist.

DR. W. W. DIMOCK (Corn. '05), of Lexington, Ky., is convalescing from an attack of pneumonia and pleurisy. He was taken sick on September 26, went to the hospital two days later and returned home on October 14.

DR. GLEN L. EBRIGHT (Chi. '08) and Mrs. Ebright, of Hammond, Ind., celebrated their 25th wedding anniversary on October 4, 1938, at the Woodmar Country Club, near Hammond. About 100 guests were present to felicitate the popular couple.

DR. M. J. EGGERT (O. S. U. '33), who has been on meat inspection service in the U. S. Bureau of Animal Industry, at Baltimore, Md., has resigned. He will be associated in practice with Dr. Edward E. Thompson (Iowa '34), at Roanoke, Va.

DR. W. S. GOCHENOUR (U. P. '13) has resigned his position in the Pathological Division of the U. S. Bureau of Animal Industry, effective November 1, to become Director of the Biological Laboratories of Pitman-Moore Company, at Zionsville, Ind.

DR. ERWIN JUNGHERR (Vienna '22), of the Department of Animal Diseases, Storrs (Conn.) Agricultural Experiment Station, has been granted a six-month sabbatical leave of absence, effective Sept. 15, 1938, for study in the Department of Pathology, College of Physicians and Surgeons, Columbia University.

DR. W. J. ANGERER (K. S. C. '33), of Atkinson, Ill., was the victim of a peculiar accident the latter part of September. As he was driving his car to make a call, he hit a bump in the road. He was thrown to the top of the car and simultaneously a bottle of medicine in the car became dislodged and fell on the seat. Dr. Angerer landed on the bottle when he returned to the seat, seriously injuring the lower end of his spine.